THE Journal of the Kansas Medical Society

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OCTOBER
1958

VOL LIX
NO X

OCT 17 1958

HIGHLY EFFECTIVE CYCLIC THERAPY

N RLUTIN®

(norethindrone, Parke-Davis)

In gynecological disorders amenable to progestational therapy, clinical effects of injected progesterone can now be produced by small oral doses of NORLUTIN. In amenorrhea, for example, 10-20 mg. daily for 5 days—after estrogen priming—will induce "...a prompt temperature rise and withdrawal bleeding 24-72 hours after medication is stopped."[1]

CASE SUMMARY[2] Amenorrhea of four years' duration in a 24-year-old married woman. A course of 10 mg. NORLUTIN, twice daily for five days, was followed after three days by menses. When no spontaneous menstruation occurred during the following 35 days, this treatment was repeated and again induced menses. Using ethisterone, similar results were unobtainable in this patient.

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PACKAGING: 5-mg. scored tablets, bottles of 30.

REFERENCES: (1) Greenblatt, R. H., & Jungck, E. C.: *J.A.M.A.* 166:1461 (Mar. 22) 1958. (2) Hertz, R.; Waite, J. H., & Thomas, L. B.: *Proc. Soc. Exper. Biol. & Med.* 91:418, 1956.

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with unexcelled potency
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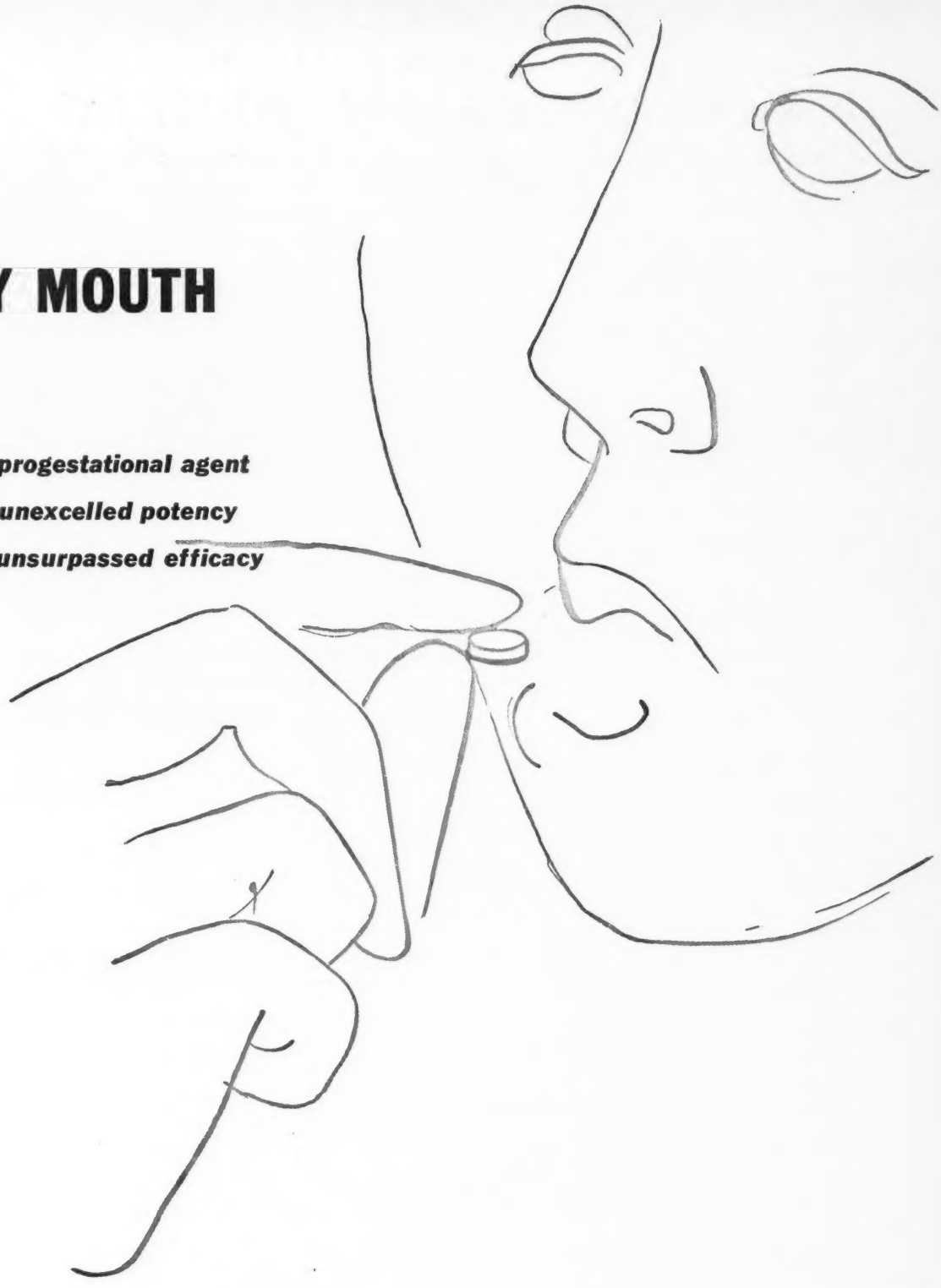

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Volume LIX OCTOBER, 1958 Number 10

TABLE OF CONTENTS

Original Articles

Oral Diuril—Study of Its Use in Treatment of Arterial Hypertension—Charles Pokorny, M.D., and L. E. Peckenschneider, M.D., Halstead 429

Evulsion of Tendons—Report of Unusual Case—Ben L. Myers, M.D., Santa Rosa, California 432

Realistic Medical Evaluations—Criteria for Employment of the Physically Handicapped—John L. Gallivan, M.D., Hartford, Connecticut 433

Leriche Syndrome—Report of Two Cases Occurring in Brothers—A. W. Robinson, M.D.; C. L. Henry, M.D.; A. V. Arms, M.D., and M. Dodge, M.D., Kansas City, Missouri . 437

Clinicopathological Conference—Acute Renal Failure, Convulsions, Hematemesis, Hypertension, and Vividialysis 446

BCG—After 35 Years—A Disaster or a Triumph?—Francis M. Ikezaki, M.D., Honolulu 460

Editorials

Radioactive Contamination 441

Profile of the Practicing Physician 441

Nutrition and a State Medical Society . . . 444

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why all the fuss over potassium?

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Dosage should be individualized.

For clinical supply and literature, write Dept. 43A

1. *Friedlander, H. S.: The role of ataraxics in cardiology. Am. J. Card.* **1**:*395, March 1958.*
2. *Shapiro, S.: Observations on the use of meprobamate in cardiovascular disorders. Angiology* **8**:*504, Dec. 1957.*

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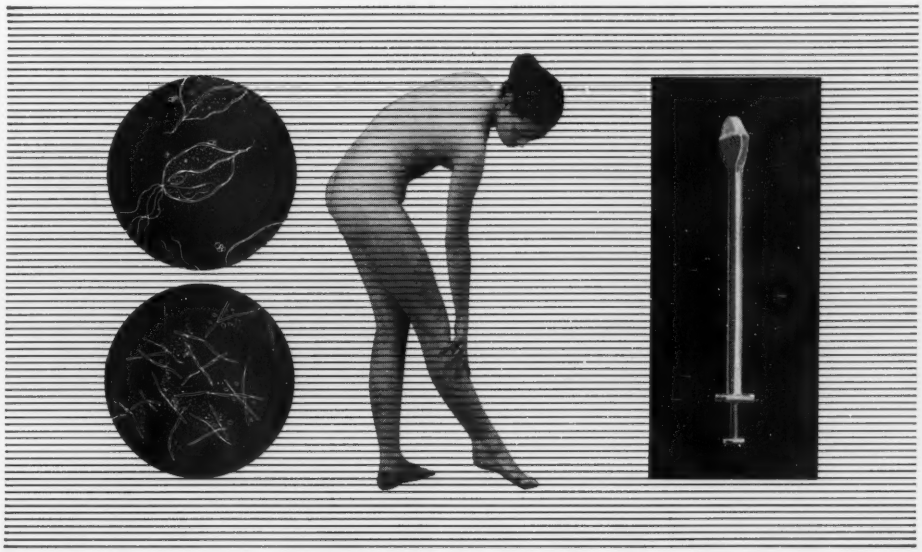

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1. Davis, C. H.: Trichomonas Vaginalis Infections: A Clinical and Experimental Study, J.A.M.A. *157*:126 (Jan. 8) 1955.
2. Pitt, M. B.: Leukorrhea, Causes and Management, J.M.A. Alabama *25*:182 (Feb.) 1956.
3. Lang, W. R.: Recent Advances in Vaginitis, Philadelphia Med. *51*:1494 (June 15) 1956.

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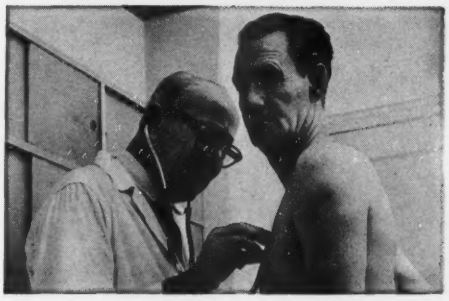

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2 to 6 years	3 cc.	
adults and children over 6 years	1 teaspoon (5 cc.)	

Supplied:
fruit-flavored, clear green syrup in 30 cc. dropper bottles.

References: 1. Drugs of Choice 1958-1959, St. Louis, C. V. Mosby Company, 1958, p. 347.
2. Goldsmith, J. W.: Minnesota Med. *40*:99 (Feb.) 1957.

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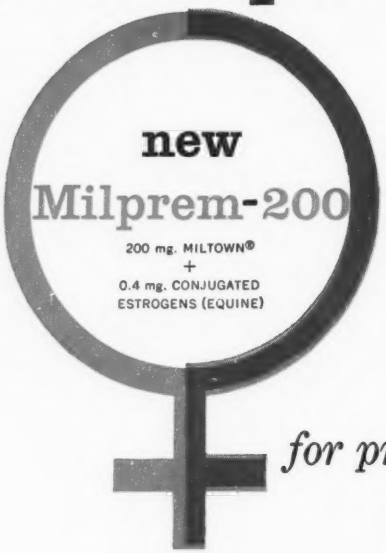

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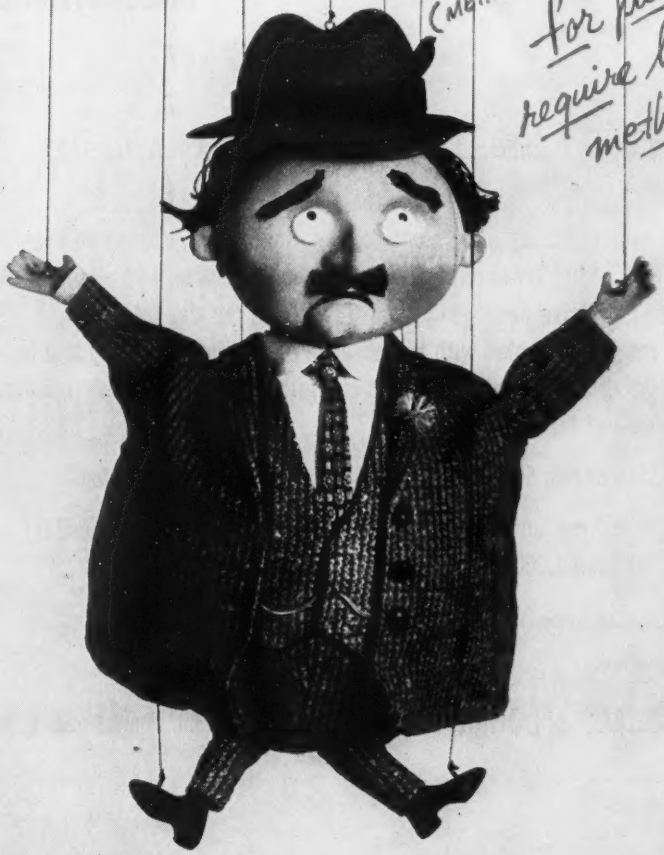

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FINNERTY, F. A., Buchholz, J. H. and Tuckman, J.: J.A.M.A. **166**:141, Jan. 11, 1958.

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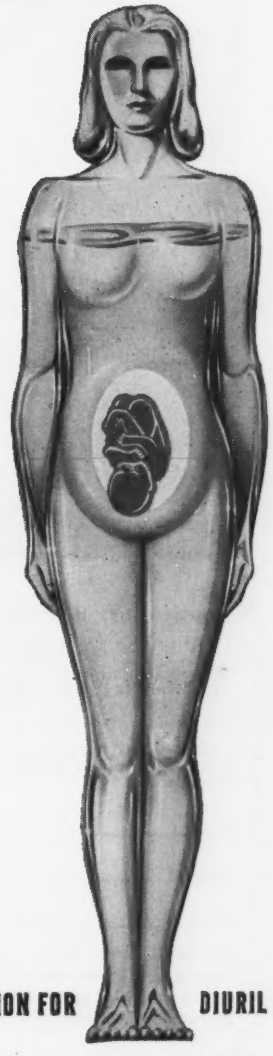

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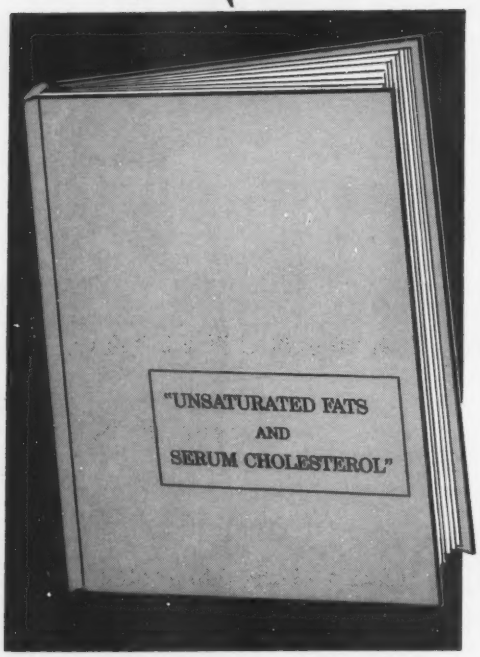

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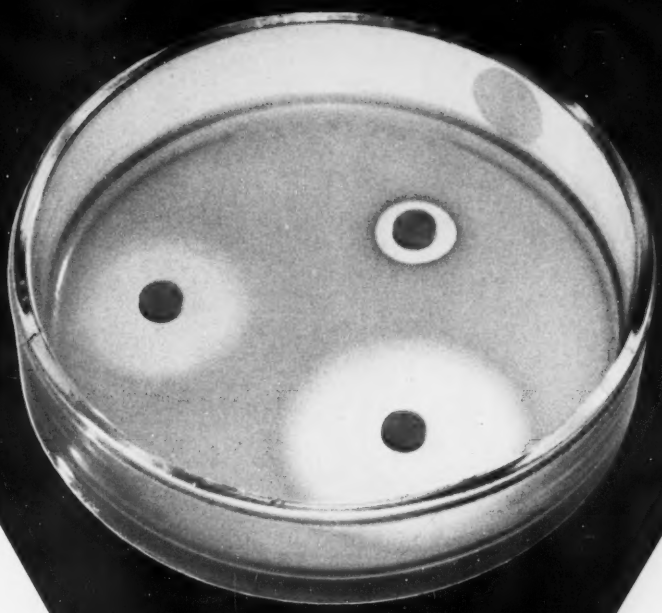

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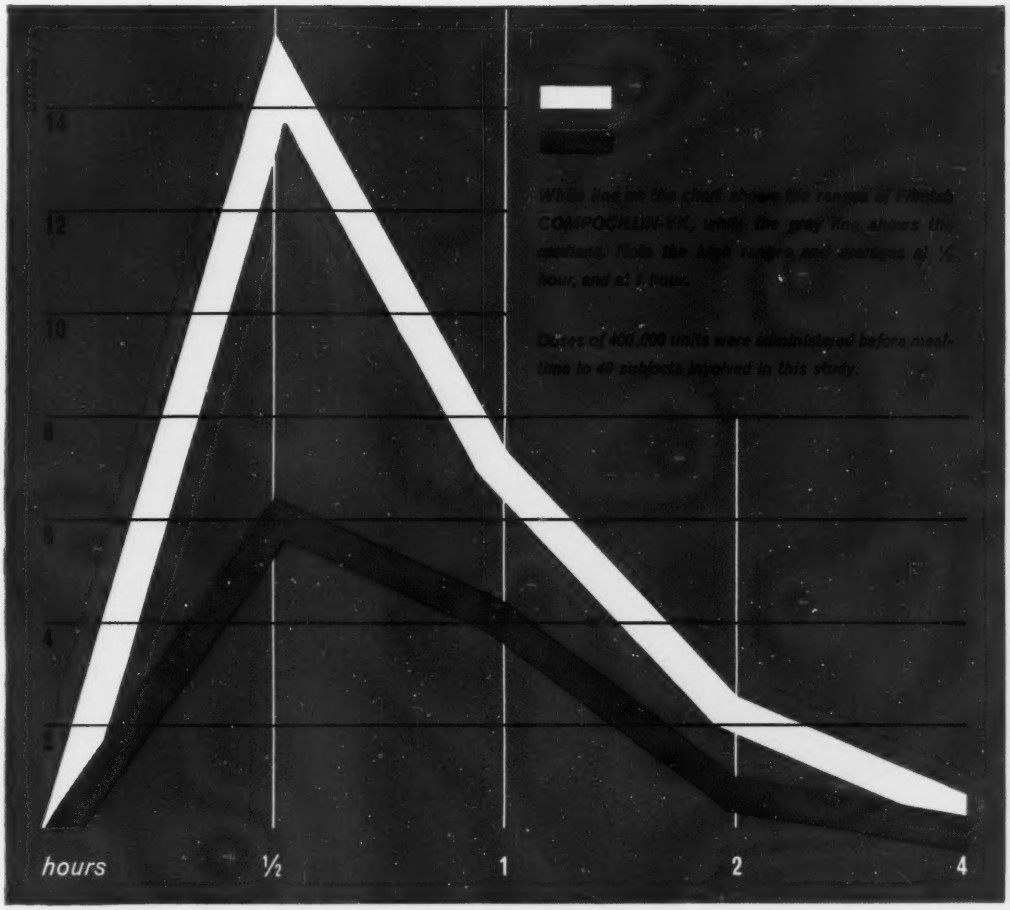

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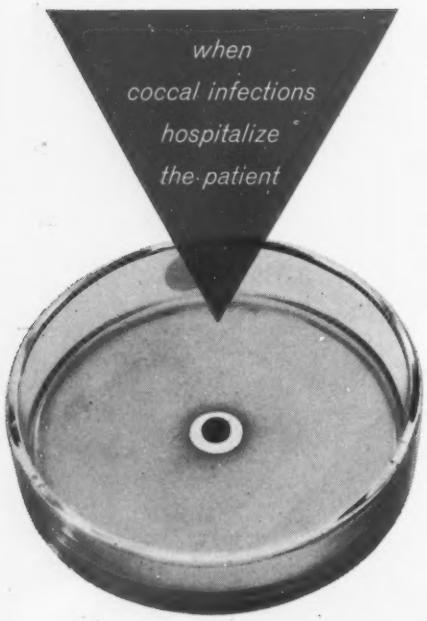

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Against a wide range of staphylococcal, streptococcal, pneumococcal and enterococcal infections. A drug of choice for treating serious infections caused by organisms that resist all other antibiotics.

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provides bactericidal action

against coccal infections

provides successful short-term therapy

against endocarditis[1]

provides clinical effectiveness against

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1. Antibiotics Annual, 1956-'57, p. 706.
2. Antibiotics Annual, 1957-'58, p. 180-7.

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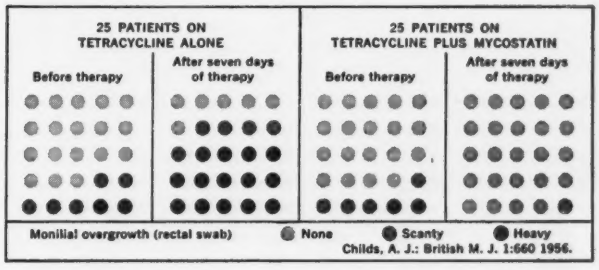

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Boron	0.1 mg.
Cobalt	0.1 mg.
Fluorine	0.1 mg.
Iodine	0.2 mg.
Magnesium	3.0 mg.
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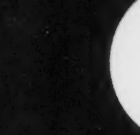

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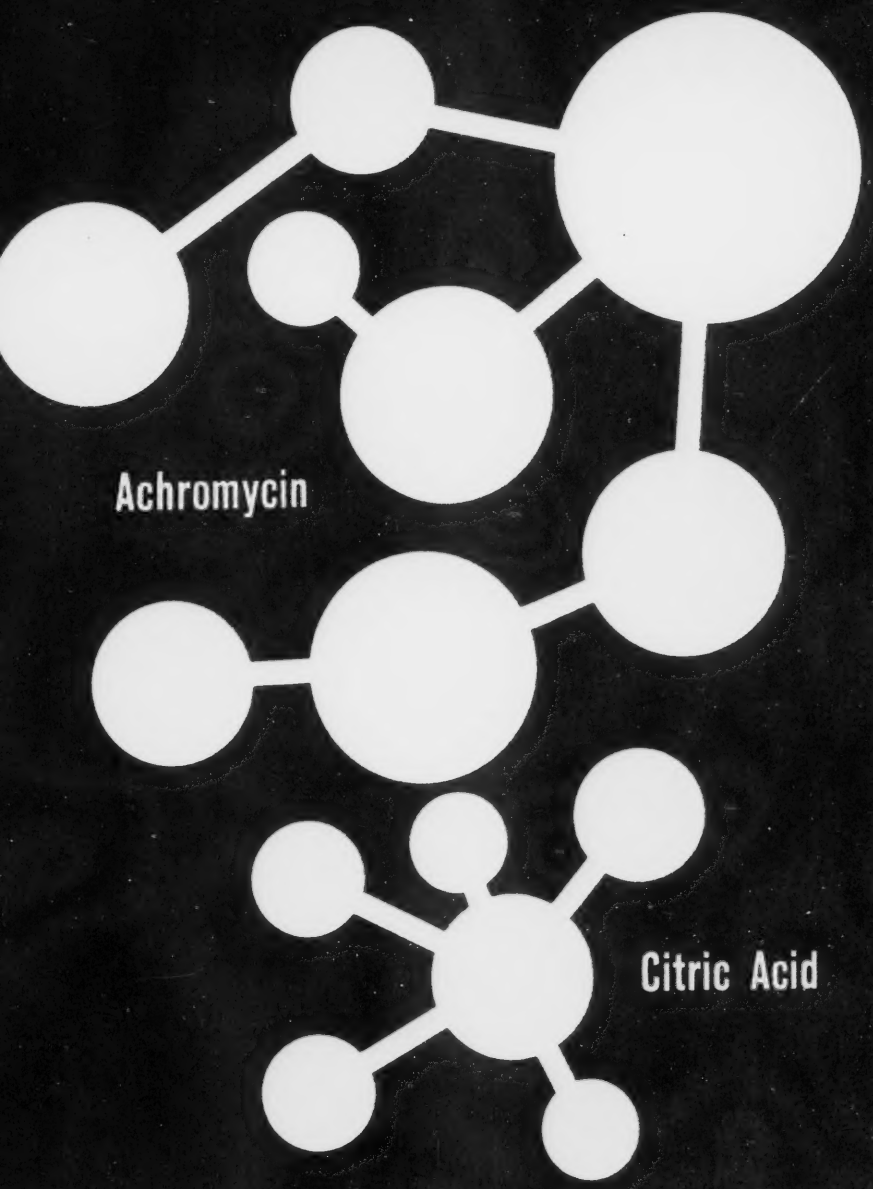

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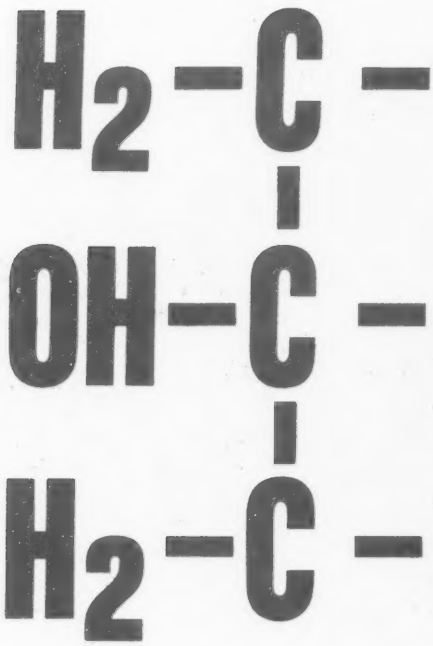

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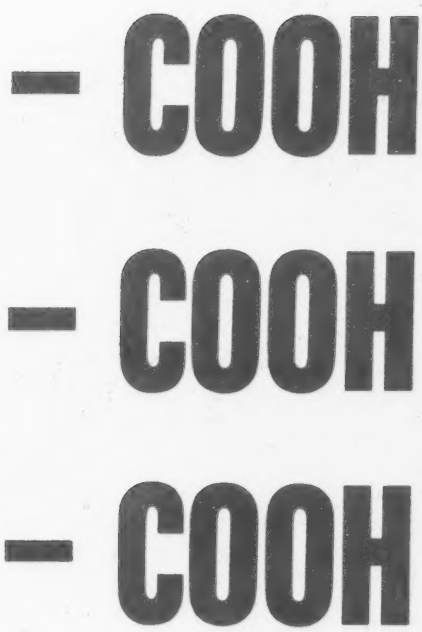

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(1) Marangoni, B. A.: Am. Pract. & Digest Treat. 8:1959, 1957.
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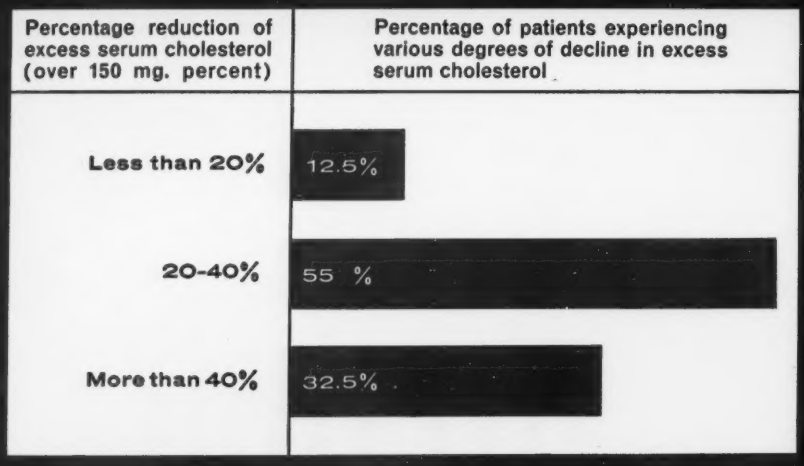
Percentage reduction of excess serum cholesterol (over 150 mg. percent)
Percentage of patients experiencing various degrees of decline in excess serum cholesterol
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12.5%
20-40%
55 %
More than 40%
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'Cytellin' provides the most rational and practical therapy available. Without any dietary adjustments, it lowers elevated serum cholesterol concentrations in most patients.

In a number of studies, every patient who co-operated obtained good results from 'Cytellin' therapy. On the average, a 34 percent reduction of excess serum cholesterol (over 150 mg. percent) has been experienced.

In addition to lowering hypercholesteremia, 'Cytellin' has been reported to effect reductions in C/P ratio, S_f10-100 and S_f12-400 lipoproteins, "atherogenic index," beta lipoproteins, and total lipids.

May we send more complete information and bibliography?

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Volume LIX **OCTOBER, 1958** **No. 10**

Oral Diuril

Study of Its Use in Treatment of Arterial Hypertension

CHARLES POKORNY, M.D., *and* L. E. PECKENSCHNEIDER, M.D., *Halstead*

Many methods have been used in the treatment of hypertension, from the marked low sodium diet, as exemplified by the rice diet, through the use of various drugs, to the surgical procedures such as splanchnicectomy. These have also been used in various combinations. With our group the alseroxylon fraction of Rauwolfia, either alone or in combination with Veratrum, has been used in the treatment of hypertensive patients.

In most instances the restriction of sodium has had an added effect. In patients with more severe hypertension who have not responded to this regimen, hydralazine hydrochloride or ganglionic drugs, or both, have been used. Most recently we have used mecamylamine hydrochloride with good effect. Problems of side effects of the ganglionic blocking drugs have been common to all, namely orthostatic hypotension, neurogenic bladder, and obstipation, although good therapeutic effect has been obtained from use of the drugs in hypertensive patients.

Recently, in an excellent paper by Hollander and Wilkins,[1] the use of chlorothiazide in the treatment of hypertension is discussed. Their results show that chlorothiazide in the human has a definite effect on electrolyte and water metabolism, decreasing the total body sodium and fluid volume, which has a hypotensive effect. Fries et al.[2] also feel Diuril appears to be a specific for the hypertensive state.

Chlorothiazide is commercially available as Diuril and is chemically known as 6-chloro-7-sulfamyl-1, 2,4-benzothiadiazine-1,1-dioxide. This is a substituted benzothiadiazine compound to which a free sulfamyl group has been introduced. In both animal and clinical observations it is shown the predominant influence is on renal excretion, on sodium, and chloride, with a similar but lesser effect on potassium and bicarbonate.[3, 4]

With oral administration the action starts in approximately two hours and lasts from 6 to 12 hours. This allows the drug to be given early enough in the day to avoid interrupting the patient's sleep by its diuretic effect. In most hypertensive patients there is an initial diuresis and a small weight loss which the patient gradually regains. After this there is no diuresis of importance.

All of our patients were on previous anti-hypertensive therapy. Chlorothiazide was added to the regimen, 0.5 gm. at 8 a.m. and 2 p.m. An average blood pressure of 182/104 was reduced to 138/84. In some patients on ganglionic blocking drugs it was found the dosage had to be reduced rather rapidly as the patient developed marked orthostatic hypotension. Heider et al.[5] have shown that chlorothiazide accentuates the effect of ganglionic blocking agents. With reduction of these agents, obstipation and symptoms of neurogenic bladder disappeared. At times the dose of Rauwolfia and Veratrum could be reduced and in many the chlorothiazide could be reduced to 0.25 gm. twice daily. Most patients commented they felt considerably better physically after several weeks on chlorothiazide.

While it has been shown that there is electrolyte

Chlorothiazide, given orally in combination with other antihypertensive drugs, has been found to be effective in treatment of arterial hypertension. A summarized report is given for 34 patients.

MEDICATIONS USED AT TIME DIURIL WAS STARTED

	Patient		Sex	Race	Age	Date	Blood Pressure	Rauwolfia	Rauwolfia & Veratrum	Apersoline	Ganglionic Block	Date	Blood Pressure	Rauwolfia	Rauwolfia & Veratrum	Apresoline	Ganglionic Block
VB	No. 1		M	C	59	11- 7-57	170/100		X		X	2-18-58	132/80		X		
WB	No. 2		M	W	54	11-10-57	184/86		X	X	X	3-16-58	154/82		X	X	
CW	No. 3		M	W	63	11- 7-57	184/94		X	X	X	4- 4-58			X	X	
AB	No. 4		M	W	43	12- 5-57	154/100		X			1-24-58	130/68		X		
PB	No. 5		M	W	67	12- 6-57	190/90		X			12-20-57	146/86		X		
JH	No. 6		M	W	63	10-30-57	200/100		X		X	2-10-58	146/84		X		
HH	No. 7		M	W	59	11-14-57	204/130		X		X	1-22-58	160/96		X		
JM	No. 8		M	W	61	1-24-58	204/104		X		X	2- 7-58	132/88		X		X
AM	No. 9		M	W	47	12-12-57	172/112		X			1-23-58	138/92		X		
ER	No. 10		F	W	45	12-11-57	166/102		X			1-27-58	134/92		X		
CW	No. 11		F	W	51	12- 4-57	210/110		X			2-10-58	134/82		X		
TN	No. 12		M	W	60	10-18-57	180/120		X	X		1-10-58	132/88		X	X	
LK	No. 13		F	W	43	1- 2-58	210/130		X			2-19-58	146/104		X		X
AA	No. 14	(A)*	F	W	53	11-25-57	194/92		X		X	2-15-58	158/80		X		
MS	No. 15		F	W	56	11-25-57	174/86		X			2-12-58	122/70		X		
SU	No. 16		M	W	62	2- 4-58	170/110		X			2-21-58	136/96		X		
AW	No. 17		F	W	74	11-10-57	190/70		X			2-16-58	168/72		X		
CG	No. 18		F	W	60	12- 9-57	174/110	X				2-13-58	132/86	X			
SG	No. 19		F	W	66	12- 9-57	200/112	X				2-28-58	150/84	X			
LH	No. 20		M	W	54	1- 3-58	185/130		X			2-24-58	140/98		X		
GG	No. 21		M	W	73	2-25-58	200/106		X			3-25-58	120/74		X		
LR	No. 22		M	W	53	12-17-57	170/100		X			3-25-58	130/70		X		
MF	No. 23	(B)*	F	W	54	9-25-57	250/150		X		X	11-25-57	142/88		X		
RM	No. 24		F	W	61	2- 7-58	180/110		X			2-21-58	126/78		X		
MB	No. 25		F	W	63	1-31-58	180/110		X			2-21-58	138/90		X		
CH	No. 26	(C)*	M	W	61	8- 5-57	164/100		X			2-17-58	130/86		X		
RD	No. 27		F	W	65	9-20-57	176/80		X			1-28-58	120/68		X		
JJ	No. 28		F	C	41	11-11-57	178/110		X			1-14-58	134/82		X		
CG	No. 29		M	W	79	10-16-57	170/80	X				2- 5-58	148/78	X			
EB	No. 30		F	W	57	12- 2-57	195/95		X		X	2-24-58	158/96		X		
EF	No. 31		M	W	75	12- 8-57	190/100		X			3-27-58	150/90		X		
JM	No. 32		M	C	64	3- 8-58	230/120		X			3-29-58	130/80		X		
LM	No. 33		F	W	64	12-16-57	196/110	X				2-26-58	124/76	X			
WF	No. 34		F	W	46	10-29-57	170/108		X			2-17-58	144/94		X		

A. Patient No. 14 developed purpura after 42 days of taking Diuril, 0.5 gm. b.i.d. There were no abnormal blood findings. The purpura cleared up after the drug was discontinued. Again Diuril was started, only to have purpura develop again. Although the hypertension was well controlled with added Diuril, the patient has refused to start the drug again.

B. Patient No. 23 had a right hemiplegia at the time Diuril was started. Rauwolfia, Veratrum, and ganglionic blocking agents in combination did not control her hypertension. The blood pressure has been well controlled since Diuril has been added, and the ganglionic blocking agent has been discontinued.

C. Patient No. 26 has been controlled with Diuril 0.5 gm. at 8:00 a.m. and 2:00 p.m. three days a week.

reduction with chlorothiazide, at the dosage we used we have encountered no difficulties. The patients have been placed on a moderately low sodium diet, and we feel that most patients have exceeded the amount allowed.

To date we have not used Diuril alone in the treatment of hypertensives. Grollman,[6] in a recent communication, suggests that this drug may lower the body sodium sufficiently to control most patients with benign hypertension. Animal experiments to be published tend to confirm this suggestion.

Side effects have been minimal. Most patients tolerate chlorothiazide well. In a previous paper we reported one patient who refused the drug after taking one gram.[7] Occasionally we have seen nausea, weakness, and mental depression. After stopping the drug for several doses, we could resume administration of the original dose with no intolerance. It has been reported that serum potassium was low in patients with similar symptoms.[1] Others have reported the major side effects are those of potassium depletion and nitrogen retention, especially in larger doses.[8] Erythema which necessitated stopping the drug has been reported. Flushing, muscle cramps, paresthesias, and lightheadedness have been reported.[1] To date no hematopoietic, hepatic, or central nervous system toxicity has been noted.

Chlorothiazide has proved to be an effective hypotensive agent in combination with other antihypertensive drugs. It is effective orally. Its toxicity is minimal. Blood electrolytes should be examined periodically since it has been shown the drug is capable of causing changes in these ions. It has been shown to be an effective saluretic and diuretic, useful in management of congestive heart failure as well as in other conditions causing abnormal retention of fluid.

Hertzler Clinic and Research Foundation
Halstead, Kansas

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THE THINKER

They may hold him in scorn and derision,
For they sense not the depth of his thought,
But they know that from out of his vision
All achievements of time have been wrought.
In his mind lies the infinite power
That can give to thin fantasy birth,
And we know that the thinker shall tower
O'er the lords and the princelings of earth!

—Roland Eberhart

Evulsion of Tendons

Report of Unusual Case

BEN L. MYERS, M.D., ***Santa Rosa, California***

Although surgical literature records an abundance of material concerning injury to tendons and improved techniques in caring for such injuries, there is little or no mention of evulsion of tendons in human beings. I have conferred with surgeons with wide experience in handling destructive injuries, with industrial physicians, and with orthopedists without obtaining information on the subject. It appears that incidents of evulsion of tendons are either not recorded or are exceedingly rare.

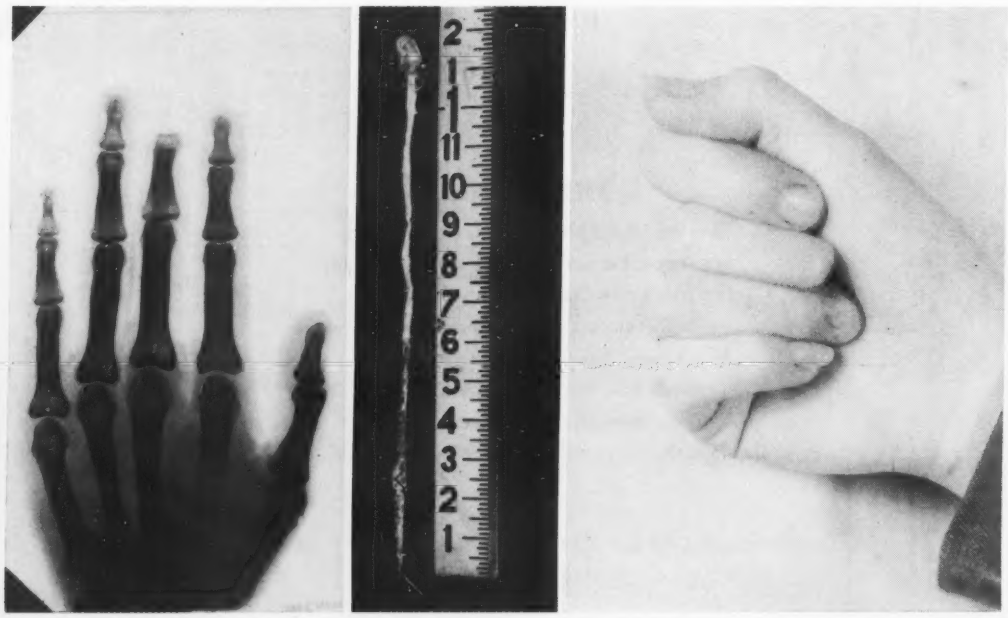
On April 11, 1945, I was called to St. Mary's Hospital, Kansas City, Missouri, to care for a 19-year-old woman, operator of a punch press, who had suffered an injury described by the house physician as follows: "The second finger was disarticulated at the terminal articulation. The terminal phalanx with the entire tendon of the flexor profundus digitorum was detached."

The author, who formerly lived in Iola and in Kansas City, Missouri, is a member of the Allen County (Kansas) Medical Society.

Inspection showed that the punch press had traversed the terminal articulation of the second finger on the right hand with almost no injury to articular surfaces but severing all soft tissues except the flexor profundus digitorum tendon and the structure external to it on the flexor surface.

Evulsion of tendons either occurs rarely or is not reported since surgical literature carries little mention of the subject. A case from the author's experience is reported.

When the accident occurred the patient, being frightened, jerked. The machine held the tendon in its grasp, 13 inches of tendon being stripped from the muscle and drawn from the arm, hand, and finger. The tendon was still attached to the terminal phalanx of the finger.



At left, x-ray of injured hand. Center, photograph of specimen beside a ruler. Right, photograph of hand after healing.

The finger was given an antiseptic dressing, and the patient was hospitalized for observation and immediate treatment should an infection ensue. No complications developed. The patient had good use of her hand and was dismissed on April 20. She was followed as an out-patient through May and June.

A pathological study, made by Dr. Albert E. Upsher, was reported as followed: "A single specimen is submitted for examination which is identified as the tip of the middle finger. It possibly has been removed traumatically at about the junction of the middle and last or third phalanx. Attached to the tip of the finger, which includes the nail, nail bed, and adjacent tissues, is a tendon which measures 13½ inches in length. It is perfectly clean all the way up to presumably the attachment at the base of the muscle, where there are a few reddish fibers of striated muscle group noted. This presumably is the tendon of the flexor profundus digitorum. No sections taken. Specimen to be saved."

The radiologist who made an examination, Dr. E. R. Deweese, gave this report: "The examination of the right fingers reveals a loss of the bone and soft tissues of practically the entire distal phalanx of the right middle finger, only a small residue of the base of the distal phalanx remaining and articulating with the middle phalanx. No fractures of the bones of the middle finger or any dislocations. The ring and index fingers included in the examination showed no abnormalities."

1831 Robin Hood Lane
Santa Rosa, California

Realistic Medical Evaluations

Criteria for Employment of the Physically Handicapped

JOHN N. GALLIVAN, M.D., ***Hartford, Connecticut***

One of the predicaments we encounter in our efforts in behalf of handicapped individuals is the habit of tagging such individuals with some sort of medical or quasi-medical terms. The tag may be cardiac, epileptic, amputee, blind, deaf, diabetic, or various combinations of these and other medical or quasi-medical terms. Then, in the face of prejudices which such tags commonly call forth in the minds of laymen and sometimes even of physicians, we ask employers to hire on the basis of abilities and not disabilities. The best method of avoiding this predicament is for physicians and their assistants to keep their medical diagnostic tags to themselves, for their own uses, and to report the results of their evaluations in terms of precisely what efforts the individual can safely perform and what environmental exposures he can withstand.

Someone once said that success in industry requires the proper blending of men, materials, and machines into a profitable productive relationship with a minimum of waste, wear, and weariness. In a competitive free enterprise system, it can also be said that success depends upon building and maintaining an effective work force through making the fullest use of available skills. This in turn depends upon an effective placement program which aims to have every worker

The program of evaluation outlined here does not focus upon persons with physical impairments which can be labeled with medical or quasi-medical tags. Rather it focuses on skills, aptitudes, interests, and similar traits and is geared to the presumption that all reasonable means possible will be utilized to make productive use of those skills. In its day by day efforts to adapt the worker to the job or the job to the worker, it permits absorption of the physically handicapped into the production program as a matter of course.

Editor's Note. Although this article was prepared in a style and format suited for consumers of occupational medical services, it will be of value to readers of the JOURNAL in delineating various factors which influence evaluations. Dr. Gallivan is medical director, United Aircraft Corporation, East Hartford, Connecticut. He presented this address at the annual meeting of the President's Committee on Employment of the Physically Handicapped, Washington, D. C., on May 9, 1958.

at all times placed on the job that allows him the best use of his skills and agrees with him physiologically and psychologically.

Selective placement calls for cooperative effort, combining the quite dissimilar skills and disciplines of the social, biological, and physical sciences. The examining physician is part of this placement team, and the closer he blends his efforts with the rest of the team, the more he and that entire team will come to acquire a new point of view which is not basic to the original training in the respective fields.

The physiological evaluation of an individual comes within the province of the physician and his medical co-workers and cannot be successfully handled by persons without medical qualifications. Many purposes can be served by the medical examination and discussion of the findings by the physician with the examinee; but—from the placement standpoint—the medical evaluation should be on the basis of functional capacities and the findings should be expressed in a clear, concise description of just what efforts an examinee can safely perform and what environmental exposures he can withstand.

There are all sorts of functional capacity report forms which can be used. Basically they call for answers to questions such as:

How many hours per workday can the examinee bear the body weight, primarily on the feet in a stationary position? How many hours walking on level surfaces? How many hours going up or down stairs or inclines using the hands and arms only for balance? How much lifting, carrying, pushing, pulling, and for how long? Can he operate hand power tools? ride a tricycle? climb a ladder? be exposed to various types of oils, solvents, acids, alkalis, synthetic resins? How much noise can he be exposed to and for how long at what intervals?

These are, of course, only a few samples of the questions the physician must answer. Nevertheless, they serve to point up the fact that the physician in reporting for placement purposes should avoid medical diagnostic tags and should use numbers or precise terms wherever possible to describe capacities in lengths of time, weight ranges, distances, volumes, concentrations, etc. This is what we mean by the term "functional capacity language."

The physician learns "functional capacity language" as he blends his efforts with safety engineers, industrial hygienists, employment interviewers, foremen, and other members of the placement team. The safety engineers and industrial hygienists use exactly the same language in evaluating the physical demands and hazards of jobs.

As he works as part of the team, the physician learns more than a new language. He learns that employment interviewers and personnel investigators in a top-flight placement program will select a tentative job or occupational group on the basis of the applicant's skills, aptitudes, intelligence, interests, experience, temperament, honesty, integrity, and similar traits; but they will not concern themselves with the evaluation of the applicant's physical or physiological capacities except in two types of cases:

(1) In the case of alcoholism, narcotics addiction, sexual perversion, or similar medical conditions, the security requirements of contracts may require the personnel investigation section to reject applicants; or

(2) In cases where physiological impairments may have affected the applicant's temperament, aptitudes, interests, previous experience, etc., the employment interviewer may reject the applicant for employment without being aware that physiological impairments have been a material cause for the poor impression and record.

The physician learns further that a successful placement program is geared to the presumption that, when an applicant appears for medical evaluation after the employment office has selected a tentative job or occupational group, the employer—except in the case of the applicants who are vetoed by the personnel investigators—the employer is prepared to use all reasonable means possible to make productive use of that man and his skills. Therefore, when the man's functional capacity is found by the physician to be inadequate to meet the physical demands and environmental exposures of the tentatively selected job, additional steps are taken.

One step is to determine whether the man's capacities can be raised through medical correction or rehabilitation, through additional training, through personal constructive or protective equipment, or through other means. (An example of personal constructive equipment is a hearing aid; an example of personal protective equipment is a noise-blocker or ear defender.)

Another step is to determine whether the physical demands or hazards of the job can be reduced through operational or engineering changes in the job or whether assignment can be made to a group where teamwork can permit interchange of effort to compensate for individual deficiencies. Time spent standing, for example, is among the most frequent of the obstacles which block people from jobs; and yet analysis often reveals that much standing is simply custom and not a job essential. The reduction of lifting, carrying, pushing or pulling not only can be accomplished readily in many cases but also provides extra dividends for management when the change suggested in behalf of an impaired job candidate is found—as it so frequently is—to have application to other jobs

with resultant improvement in economy and efficiency.

Thirdly, there is the step of looking for other job openings which the applicant can fill.

All of these additional steps are greatly facilitated if the physician, in developing his medical evaluation, keeps them in mind, and in reporting his findings includes sufficient information to permit these steps to be brought smoothly into play. In cases where the applicant's functional capacity is not fully adequate to meet all the job requirements, the physician's evaluation is not complete until he has taken part in these additional steps of the employment team.

From what has been said so far, it is evident that I believe that a comprehensive selective placement program for all job applicants and employees is an essential ingredient of the success of modern industry. This program calls for a cooperative effort of persons with dissimilar training and skills. The key person in the evaluation of the physiological qualifications of the applicant or the worker is the physician. While his medical examination findings can serve many useful purposes, the physician, as part of the placement team, should develop and report his placement recommendations in "functional capacity language," and he should take an active part in the consideration of capacities-raising and demands, lowering efforts as the employment team seeks to use every means possible to make productive use of persons with useful skills. His contribution will be most effective when there is a physical demands analysis recorded for every job in the facility involved.

A program of the sort I have outlined does not focus upon persons with physical impairments which can be labeled with medical or quasi-medical tags. Rather it focuses on skills, aptitudes, interests, and similar traits and is geared to the presumption that all reasonable means possible will be utilized to make productive use of those skills. In its day by day efforts to adapt the worker to the job or the job to the worker, it permits absorption of the physically handicapped into the production program as a matter of course.

Although I have urged that the physician avoid the use of medical diagnostic terms in making reports and recommendations concerning individuals, these terms do serve a useful purpose in the development of statistical analyses carried out to measure the effectiveness of a program. For example, in the Pratt and Whitney Aircraft Division of the United Aircraft Corporation, the world's leading aircraft engine manufacturer, an analysis was made of the medical examination tabulating file as it stood on the first day of October 1956. In this analysis, a count of the hourly or shop employees according to the primary disorder, if any, showed that of a total number of 27,253 hourly or shop employees, 10,472 or 38.4 per cent had, at the time of the latest available medical examination, at least one defect of sufficient extent to cause some impairment of functional capacity. A similar study made in 1951 showed 37.5 per cent, and this proportion is typical of the past 10 years. The rate of employees in this division with medical disorders causing some functional impairment was found in studies made at various times during the 10-year period, 1948 through 1957, to run between 2¼ and 2¾ times as high among hourly or shop workers as the rate of salary or office workers.

The percentage of employees in this division with medical disorders causing some functional impairment was not the result of any concerted effort to search out and employ the so-called physically handicapped; but was the result of consistent application to all job candidates all of the time of the principles outlined above. Over the 10-year period, 1948 through 1957, excluding applicants who of their own volition did not show up to complete the employment process, 96 per cent of the applicants the employment section selected on the basis of skills and personal qualifications, other than physical qualifications, were signed into work. Over two-thirds of the 4 per cent who were not signed into work were persons rejected by the personnel investigation section. Placement failures chargeable to the health and safety section consistently ran under 2 per cent in each of the 10 years and for the entire period.

In a large facility such as this where proportionately more than twice as many shop employees show medical disorders causing some functional impairment as the salary or office workers show, a comparison of absenteeism records is of some interest. The male shop employee is absent about 25 per cent more frequently than the male office employee, and the female shop employee is absent about 33 per cent more frequently than the female office employee. In both population groups, the frequency of female absences runs slightly more than twice as high as the frequency of male absences. However, among hourly employees only 27 per cent of the absences run for more than four days; whereas, among salary or office workers slightly over 50 per cent of the absences run for more than four days. Because non-occupational sickness and accident absenteeism benefits are payable only for absences of four days or more, the average salaried employee files a significantly higher number of claims and collects a significantly greater amount of money under this program than does the average hourly or shop employee.

These facts permit no direct inference as to the rate of absenteeism of physically impaired employees as compared with the rate of absenteeism of physically

unimpaired employees; but one can say that in this particular facility the hourly population, with a much higher percentage of physically impaired workers, shows a better record with respect to non-occupational sickness and accident absenteeism insurance experience than the salary or office population with its much lower percentage of physically impaired workers.

In the case of workmen's compensation injuries, it is logically to be expected that hourly or shop employees would show a much higher rate than salary or office employees.

The cumulative lost time occupational injury frequency rate for this particular division for the 10-year period was 4.22 cases per million man-hours worked. The highest was 5.9 in 1953 and the lowest was 3.2 in 1949. A million man-hours represents about 500 man-years of work. Therefore, a lost time occupational injury rate of 4 represents one lost time occupational injury in 125 man-years of work. The occupational injury record just cited is not remarkable but it is respectable, and it was achieved in a large manufacturing facility where more than one out of every three hourly or shop employees could be classified as physically impaired.

The above figures and statements represent only a very small portion, of course, of the data which might be cited to illustrate the fact that the type of program outlined in this paper is not a theoretical approach based primarily on humanitarian considerations but is a realistic approach based on sound business considerations. If it were to be followed universally, there would be no need to use the highly ambiguous term "physically handicapped."

The final physiological evaluation of a job applicant or a worker must obviously fall to a physician. If the physician will learn some of the techniques of the specialty of occupational medicine, if he will develop and report his placement evaluation in "functional capacity language" and avoid the use of medical diagnostic tags in making placement recommendations, if he will work as part of the employment team taking part in the consideration of capacities-raising and demands-lowering efforts as the employment team seeks to use all practical means to make productive use of the man and his skills—in short, if he will follow the principles outlined above, his medical evaluation will be realistic.

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Leriche Syndrome

Report of Two Cases Occurring in Brothers

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Introduction

Since the first description of thrombo-occlusive disease of the aorta and its bifurcating iliac branches by Leriche in 1940[1] and the first homologous graft of the abdominal aorta by Oudot[2] in 1951, a surprisingly large number of cases have been surgically corrected. The occurrence of Leriche syndrome in siblings is unusual, and our recent experience with successful surgical management of this disease in two brothers seemed worthy of presentation.

Report of Case 1

H. H., a 43-year-old shoemaker, had noted progressive intermittent claudication in the thighs and both lower extremities of 11 years' duration. At the time of evaluation in July 1955, he had experienced pain on walking a distance of two and one-half to three blocks. In 1950 at age 38 he was diagnosed elsewhere as having coarctation of the aorta. In February 1955 he had suffered a cerebral vascular accident with left hemiparesis which gradually cleared. He had noted frequent occipital headaches of six years' duration.

Physical examination: The blood pressure in the right arm was 270/140 and in both legs 110/95. Pulse was 100 and regular. Funduscopic examination revealed marked A-V constriction with tortuosity and focal arteriolar constrictions. A grade II apical systolic murmur with extension over the lower sternum was present. Femoral pulsations were greatly diminished bilaterally.

Laboratory and x-ray examinations: Routine complete blood count and urinalysis were within normal limits. Mosenthal kidney concentration revealed a specific gravity 1.013 to 1.022. The electrocardiogram showed a left ventricular strain pattern. Chest x-ray was considered within normal limits with equivocal evidence of rib notching.

Hospital course: A left thoracotomy was performed, revealing a normal thoracic aorta without evidence of coarctation. It still was felt this patient suffered from aortic obstruction because of the pressure gradient between the upper and lower extremities. Therefore, following convalescence, a translumbar aortogram was performed which established the diagnosis of Leriche syndrome with complete block of the abdominal aorta just distal to the renal arteries at approximately L-1 (Figure 1). The right kidney appeared hypoplastic. Interim management of this patient consisted of hypotensive therapy, and he was released to go home with instructions to take Apresoline 400 mg. and Rauwiloid 8 mg. daily.

The occurrence and successful surgical management of Leriche syndrome in two brothers are reported. The differential diagnosis between Leriche syndrome and coaretation of the aorta is discussed.

Figure 1. Case 1, H. H., aortogram showing complete block of the abdominal aorta just distal to the renal arteries.

From Departments of Medicine and Surgery, St. Luke's Hospital, Kansas City, Missouri.

Second hospital admission: When he was readmitted to the hospital in January 1956 he continued to complain of bilateral thigh and calf claudication and reported two episodes of transient paroxysmal tachycardia. Physical examination at this time revealed the blood pressure to be 295/150 in both arms. Pulse was 120 per minute.

Laboratory and x-ray examination: Hemoglobin 15 gm., red blood count 5.3 million, urinalysis, specific gravity 1.008, albumin 1 plus, non protein nitrogen 37 mg. per cent, fasting blood sugar 104 mg. per cent, serum sodium 143 mEq./l, serum potassium 3.5 mEq./l, serum chlorides 94 mEq./l. Retrograde pyelography revealed moderate hypoplasia of the right kidney, and no indigo carmine appeared from the right ureteral orifice following intravenous injection. An electrocardiogram was interpreted as evidence of marked left ventricular strain. Repeat chest x-ray was not remarkable.

Hospital course: On February 1, 1956, exploratory laparotomy was carried out with confirmation of occlusive disease involving the terminal aorta and extending from just below the renal artery origin down to and including the aortic bifurcation. The aorta at this level was cord-like and non-pulsatile. The right renal artery was totally occluded and the right kidney was grossly contracted. A right nephrectomy was performed. Proximal and distal clamps were then placed, and resection of the terminal aorta and bifurcation were accomplished with subsequent placement of an aortic bifurcation homograft. Proximal and distal endarterectomies were performed prior to placement of the graft with satisfactory retrograde flow being established. Continuous 5-0 arterial suture was used for vascular anastomosis.

Grossly the resected aorta revealed thickened, calcific, hyalinized and dense sclerosis involving all layers. The right kidney weighed 80 grams and showed a slightly granular surface with an extremely thin cortex on section. There was an associated marked arterial and arteriolar nephrosclerosis.

During the immediate postoperative period the blood pressure ranged from 160/95 to 220/100, and bilateral femoral pulsations of good quality were present. The patient was digitalized because of persistent tachycardia and dismissed from the hospital on February 18, 1956. A follow-up visit on April 18, 1956, revealed blood pressure in the arms to be 170/100, legs 110/70. He was again seen on May 28, 1958, at which time his blood pressure was recorded as 170/90 in the arms, 110/90 in the legs. Good femoral pulsation was evident, peripheral lower extremity pulsations absent. Laboratory examination revealed normal urine, complete blood count, electrocardiogram, and chest x-ray. The previous left ventricular strain pattern had been completely reversed. He was working full time in his former job capacity. He complained only of weakness in the left upper extremity and mild blurring of vision since his cerebral vascular accident in 1955. Since his surgery he has had no further claudication.

Report of Case 2

J. H., a 56-year-old shoe factory worker, had noticed progressive intermittent claudication of the hips and thighs of five years' duration, more pronounced on the left side. Intolerance to cold was noted in both feet, again predominately on the left. He smoked one package of cigarettes daily. He had been informed elsewhere one year before that a mild elevation of blood sugar was present, and he had followed a carbohydrate restrictive diet. Impotence had been evident for approximately one year.

Physical examination: Blood pressure was 140/90, pulse 85 and regular. Funduscopic examination was negative. Marked cyanosis of all toes of the left foot and of the right first toe was present. Femoral and all peripheral pulses of the left lower extremity were absent. In the right lower extremity the femoral pulse was markedly diminished, and the lower right peripheral pulsations were absent. Marked pallor and rubor were demonstrated in the elevation-dependency test in both feet. Venous filling time in the legs was delayed, 35 seconds on the right and 40 seconds on the left.

Laboratory and x-ray examinations: Routine urinalysis and complete blood count were within normal limits, fasting blood sugar on two occasions was 154 mg. per cent and 119 mg. per cent, non protein nitrogen 38 mg. per cent, total cholesterol 197 mg. per cent, cholesterol esters 56 per cent, electrocardiogram and chest x-ray normal. Trans-lumbar aortogram revealed a complete obstruction of the abdominal aorta immediately distal to the point of origin of the left renal artery (Figure 1).

Diagnoses of thrombo-occlusive arterial disease of the aorta and mild diabetes mellitus were made.

Hospital course: On February 19, 1958, exploratory laparotomy was carried out and the abdominal aorta surgically exposed. Extensive complete occlusive disease of the aorta was encountered, extending from just below the renal arteries down to and including both iliac vessels. Proximal and distal clamps were applied, and the aorta and bifurcation were resected. Endarterectomy was carried out in both iliac vessels and the proximal aortic segment. Free retrograde bleeding was obtained from the iliac vessels.

Gross and histologic examination revealed a greatly thickened calcified atherosclerotic aorta with the lu-

men completely filled with laminated partly calcified thrombus.

Postoperatively, normal color gradually returned to the feet and legs with associated femoral pulsations bilaterally. His recovery was uneventful, and he was dismissed from the hospital on March 5, 1958. The last checkup visit on April 18, 1958, revealed normal circulatory status of the lower extremities. The patient has returned to full time work in his previous capacity.

Discussion

Both of these men with Leriche syndrome presented classic histories and findings of bilateral claudication of hip and thigh pain with associated absence or diminution of femoral pulsations. Trans-lumbar aortography firmly established the diagnosis in both instances.

It is known that atherosclerosis is predisposed to develop in certain families and that the more extensive lesions often appear at the same site. In the siblings reported here the entire lower abdominal aorta was occluded, extending proximally to the level of the renal arteries.

The Goldblatt phenomenon is well demonstrated in Case 1 (H. H.) by the remarkable drop in systemic blood pressure and clinical improvement following the removal of the ischemic contracted kidney.

The differential diagnosis between thrombo-occlusive disease of the aorta with associated hypertension and coarctation of the aorta can at times present a problem, as evidenced by the initial confusion in the diagnosis of Case 1 (H. H.). Co-existent hypertension with Leriche syndrome has been reported in from one-fourth to one-third of large case series Brachiocephalic hypertension associated with a pressure gradient between upper and lower extremities due to aortic obstruction may appear in both entities and, therefore, other criteria must be relied upon to differentiate the two. Intermittent claudication present in most patients with Leriche syndrome is recorded in five per cent of patients with coarctation. Clinical evidence favoring coarctation would include vigorously pulsating collateral vessels, interscapular systolic murmur, and x-ray findings of diminished aortic knob, rib notching, and soft tissue prominence of the left subclavian artery. Coarctation of the aorta is, of course, a congenital malformation and is usually manifest clinically within the first three decades of life, whereas acquired thrombo-occlusive aortic disease usually appears at a later time. If doubt exists as to the diagnosis or if surgery is contemplated, aortography should resolve the conflict.

Figure 2. Case 2, J. H., aortogram showing complete block of the abdominal aorta just distal to the renal arteries.

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PRESIDENT'S PAGE

DEAR DOCTOR:

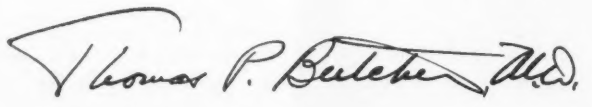
Last month Mr. Ebel and your president attended the Public Relations Meeting sponsored by the A.M.A. Mr. Leo Perlis, representing the AF of L-CIO, presented labor's views on health care. He made it quite clear that labor regards organized medicine as a doctors' labor union which exists to further the doctors' interests. This was not a criticism—it was a plain statement of what he believed to be a fact. As such, he respected organized medicine and proceeded to deal with it as a bargaining agency representing the doctors' interests.

Now this was not surprising, coming as it did from one whose life work is along such lines in behalf of labor. The thing that amazed us was that many of the others in attendance, medical society executives, etc., seemed to accept his interpretation without protest.

Perhaps your president is wrong. Perhaps the chief function of organized medicine is to serve the physician. But we will contend that organized medicine and its component physicians have always existed and do exist primarily to serve the patient and the public health.

Such service has enjoyed, and will continue to enjoy, public confidence and respect just so long as the doctors and their organizations keep it that way. In dealing with economic and political forces it behooves us to keep our public service and obligations ever in the foreground. Most of the groups that strive to preserve the American way of life represent interests that may properly be termed selfish. We, as physicians, have a unique opportunity as defenders of the faith—the faith in the brotherhood of man and the personal dignity of the individual. We must not permit our antagonists to crowd us into the corner of self interest.

Fraternally,



President

EDITORIAL COMMENT

Radioactive Contamination

Editor's Note. This editorial on protection against radioactive contamination in the laboratory was prepared by Dr. G. R. Germann, chairman of the Public Health Committee of the Kansas Radiological Society, as a follow-up on his article on radiological safety in the September issue of the JOURNAL. *Dr. Germann is a radiologist at the University of Kansas Medical Center.*

In a previous editorial we commented on general protection from radiation and how physicians can protect themselves and their patients. However, with the increasing number of isotope laboratories, certain rules for protection here would also seem in order.

If the laboratory is an active one, it seems almost inevitable that contamination will occur at some time. Therefore, the person in charge of the laboratory should be accident conscious, and his entire routine should be such that only minor contamination can occur so that control will be relatively easy. One of the easiest and most effective steps is to see that all "work-tops" are covered with absorbent disposable material. Diaper material or sheet wadding serves very well.

However, should contamination occur, it will involve the physical plant of the laboratory, or the person in the laboratory, or both. The physical area is easily decontaminated with absorbing paper followed by soap and water which is also removed with absorbent paper. It is also very helpful that the non-radioactive chemical can be used on the area of contamination. For instance, sodium iodide can be concentrated on the area where I^{131} may have been spilled, and this will aid in removal of radioactive material from most surfaces. After cleaning, the area should be surveyed with a counter.

Contamination of the skin is a more serious matter. This is also a major problem in fallout where the total body may be in contact with the radioactive material as compared to a relatively small area in laboratory accidents; here again the first step is in cleansing the skin contaminated. The use of soap and water is good, but many mild soaps do not emulsify the normal fatty material in the skin which has been contaminated. Tincture of green soap, for scrubbing, is good, but the skin should not be broken since this would facilitate absorption. Detergents or actually the waterless cleansers, such as Vibra-Clean, are probably the most thorough cleansers. Waterless cleansers would be especially valuable if there were question of water contamination in addition to skin contamination. Again, in personal decontamination, the value of the non-radioactive element as a surface absorbing substitute for the radioactive material should not be overlooked. We have seen sodium iodide do an excellent job removing I^{131} from around the base of a fingernail which had become contaminated with I^{131}.

The inhalation or ingestion of radioactive materials from the laboratory is a preventable accident. No eating, drinking, or smoking can be permitted in the laboratory at any time. The use of rubber gloves also prevents hand to mouth contamination. Frequent cleansing and good general technique are of course paramount.

Should oral ingestion occur, treatment would depend on the chemical involved. I^{131} ingestion would be treated with iodine and anti-thyroid drugs.

Prevention of accidents is always the best protection. Experience is probably the best teacher here. Certainly students must work only with low activity until they are familiar with tools and techniques.

However, accidents should not produce panic and reasonably simple methods can be used to decontaminate areas and persons.

Profile of the Practicing Physician

The Metropolitan Life Insurance Company sponsors many programs which have an indirect effect upon the health of the nation—their educational advertisements in nationally distributed magazines, their compilation of statistics on morbidity and mortality, their professional and lay publications.

An interesting example of the Metropolitan's service to medicine can be found in current issues of the *Health Bulletin for Teachers,* a publication now in its 27th year. For this school term, the theme of the pamphlets will be "Health Education and the Physician." If teachers can convey to their students the spirit emanating from such a publication, medicine will be the beneficiary. The text of the first bulletin, entitled "Profile of the Practicing Physician," is reprinted below.

"Fifty years of service to medicine! As I look back and consider other careers that I might have chosen, I sometimes feel that I would settle for a less arduous calling. But on more sober thought, I know there could be but one life for me—the life of a doctor."

These words were spoken by a physician whose name is all but unknown beyond the borders of the community which he has served for half a century. Yet, this simple quotation is an appropriate prelude to our "Profile of the Physician."

In the first place, it bespeaks the deep devotion which most physicians feel for the fraternity of medicine. In the second place, it explains why the great rank and file of doctors make medicine such a vital, comforting, and reassuring thing in the lives of the people they serve. It is your doctor and mine—using in his daily rounds the hard-won knowledge of the masters, upholding in his daily life the ethics and traditions of the healing art—who makes the *practice* of medicine so beneficent a science.

What, then, of the true physician—his skills, his ideals, his ethics? How does he come by them? What personal traits make him worthy of the title, Doctor of Medicine? What new missions beckon him? And, finally, what are his rewards?

The Making of a Physician

Sir William Osler, that immortal physician, once said that "work is the master word in medicine." It begins on the very first day of the doctor's training and it increases each day thereafter. Preparation for the practice of medicine, perhaps more than for most other human undertakings, is a combination of unending work and study.

Various experiments have been tried to make the academic training of physicians less burdensome. But these plans have only demonstrated that there can be no shortcuts in the making of a physician. In fact, authorities generally agree that the long and difficult years of preparation—including two to four years of premedical study—are necessary if we are to continue to have physicians educated in the best traditions of both the humanities and the sciences.

Actually, education for a scientific career may well begin in childhood when a youth becomes absorbed in the world of growing things. Or, it may start in a high school biology laboratory when the wonders of the more primitive forms of life unfold before youthful eyes. If would be a splendid thing if all of us—especially parents and teachers—could detect the very earliest stirrings of scientific curiosity in the young mind and encourage them from the outset.

Fortunately, the "raw materials" of which physicians are made are sometimes plain to see. We should watch for a genuine interest in "the how" and "the why" of things. We should watch, too, for a more than ordinary curiosity about the structure and functions of the human body. (What makes blood red? What makes the heart beat?) And we should be alert to the young person whom others of the same age instinctively trust. A noted medical educator has cited this as "a good sign of the budding doctor."

As to the physician's formal education, most medical schools still divide the four-year course into preclinical and clinical halves. The first two years of the prospective doctor's studies center upon the normal structure and functions of the human body and the diseases that beset it. These are the years of books and microscopes and laboratory experiments devoted to learning the architecture of the body and its "biological wisdom." There is an increasing trend in many schools, however, to provide students from the start of their training with experience in family and community health problems.

In the last two years, the student passes from the classroom to the hospital wards and clinics where he begins to breathe the atmosphere he has longed for. There, under the guidance of professors skilled in various specialties, he examines and treats patients—and sharpens his powers to observe and deduce, sometimes with the aid of diagnostic instruments, and always with eyes, ears, and hands which will become increasingly sensitive to manifestations of health and disease.

Although the medical student becomes well versed in the marvelous complexity of the human body and its responses to an incredible number of insults, the aim of the medical school is not to produce a full-fledged doctor. Nor do one or two more years of hospital internship following graduation produce the complete physician. Indeed, his schooling and hospital experience give him only a good grasp of the fundamentals of medicine. And upon this basic knowledge he will build for the rest of his days because medicine, like so many other fields, is never static.

The Motives for Medicine

Medicine is no easy path to great riches. The motivations for embarking on a career in medicine are many. Perhaps one derives from an inborn love of science as applied to life and health. And the person who experiences this love simply cannot contemplate any other mission. Medicine may lure a few who see in it the opportunity to win fame by doing the spectacular—to performing seemingly impossible operations, for example, or discovering the cure for cancer. Many medical careers are also shaped by family traditions or by the desire to follow in the footsteps of a physician whom one has admired. Though the motivations for choosing medicine as a career do vary, the best medical students and the best doctors are surely those who have a genuine interest in science, an equally genuine interest in people, and a sincere desire to serve their fellow men.

The Character of the Physician

While education, training, and experience are basic requirements for a well-rounded physician, certain

personal qualifications for the profession are no less important. Dr. James A. Miller has written: "There is need for those other factors which are moral, ethical, or even spiritual to complete the equipment of the true physician. These factors are the imponderables, but they are none the less the essentials. They determine the quality of the individual in relation to his patients, to his fellow practitioners, and above all to his own inner self."

Since medicine was born of the sympathy of man for man, the physician if he is to be a true physician, should have a great capacity for sympathy, the courage to act when delay could mean the difference between life and death, the ability to influence people for their own good, and a reverence for life. In addition, the physician needs good judgment, patience, optimism, equanimity, honesty and, most important, a warmth and tenderness of personality that will bring confidence and calm to distraught individuals. Dr. Hans Zinser observed that "when such qualities come together in an individual who chooses the calling of Medicine, then we have the great physician."

The Physician's Ethics

More than three centuries before the birth of Christ the physician's way of life was spelled out in the Oath of Hippocrates. The maxims and ideals of conduct which this noble document specified are still subscribed to by physicians all over the world. It could serve as a guide to good conduct in all walks of life. To cope with changing times and conditions, physicians have also established "codes of ethics." In the medical sense, ethics is defined as "the science of values in relation to the conduct of life as a whole." Among many items found in a recent ethical code of the American Medical Association, one of the most altruistic is that during disasters and epidemics the physician will continue his work without regard for his own health or safety. It is significant that the oaths, codes of ethics, and rules of conduct to which physicians are bound are formulated primarily for the welfare of patients and secondarily for the continuing good name of the medical profession.

How the Physician's Practice Changes

As the world changes, so does the practice of medicine. Years ago, the physician was primarily a family doctor possessed of deep understanding and practical wisdom, but with no more than a handful of really effective weapons against disease. Now the doctor has a wealth of amazingly effective therapeutic agents and improved techniques for the diagnosis, prevention, and treatment of diseases.

It appeared for a time that rapid strides in the science of medicine would submerge the ancient art of the physician. Fortunately, however, the art of medicine has been saved by the physicians themselves, because they realize that they need to know people and their worries and problems almost as well as they know physical signs and symptoms. Dr. Harvey Cushing underscored this years ago when he said: "A warm, friendly relation with a patient may often yield information more valuable to the physician than anything revealed by a laboratory test."

To bring the art of the physician to its fullest flower, many medical schools now assign their first-year students to small groups of patients selected for family care. The student is introduced as a doctor in training to "his family," the patients are informed that he will *not* treat them, but will act as an advisor on problems of health. Thus, the student learns at the outset of his medical career that illness can be caused or complicated by worries over the family budget, the morning's headlines, or whether Johnny will make it through high school.

Progress in medicine as a science has, however, brought about many changes in the physician's practice. Since no one man can master the whole of modern medicine, specialists have come to play a larger part in the care of the sick. There are at present 15 or more "Specialty Boards" to certify physicians capable of pursuing the specialty of their choice. To be eligible for a specialty certification, the physician must be a graduate of an approved medical school and must have had at least five years of postgraduate training and experience before admission to a rigid written and oral examination. The specialist, in cooperation with the general practitioner, has greatly improved the treatment of many illnesses.

Physicians are making increased use of hospitals where they find a concentration of skills, special knowledge, and facilities available for the benefit of patients. Medicine, then, is becoming more and more a matter of teamwork.

Greater freedom from many time-consuming practices of the past allows the physician of today to devote more of his efforts to preventive medicine. In fact, more people than ever before are turning to him for health counseling. We look to him for advice on nutrition, exercise, rest, relaxation, accident prevention, and other matters that affect health. Mental and emotional disorders which were once either endured or charged off to personal peculiarities are now brought to the physician's attention far more often than in the past.

New Roles for Physicians

The physician is no longer "isolated on an island of disease with his patients." His experience and knowledge are being brought to bear on a wide vari-

ety of affairs ranging from social issues at the community level to matters that are international in scope. A call to physicians to exert themselves for the betterment of their communities was recently sounded by Dr. David B. Allman, President of the American Medical Association. He urged them to abandon "the ivory tower of professional detachment" and step more actively into community affairs. It is interesting to note in this connection that physicians of earlier times played prominent roles in many activities beyond the scope of medicine. For example, among those who signed the Declaration of Independence, five were physicians.

Evidence of the physician's expanding role is seen almost everywhere—in public health movements, in the formulation of laws to safeguard health, in the promotion of improved social services, in the progress of industrial health, to mention a few.

Physicians are proving themselves to be effective ambassadors of good will for the United States and Canada in foreign lands. Since disease knows no bounds, the physician and his co-workers are "ambassadors" whom we can send forth without doubt that they will be acceptable. This has already been demonstrated by the World Health Organization in its programs for malaria control, better nutrition and sanitation, health education, and accident prevention.

We live at a time when some new and exciting chapters are being added to the history of the human race. For instance, we are now well into the Atomic Era and the Age of Space is dawning. Both of these new ventures pose problems to challenge our best medical minds. Our health and safety in an atomic world may well concern the physician no less than the communicable and chronic diseases with which he has long dealt. Indeed, the physician already occupies a key position in the public health aspects of atomic energy. And the conquest of space will depend largely on the solution of problems in human physiology and psychology. The physician's contributions to space medicine may be of immediate practical value to doctors in current practice since so much is being learned about how the human body responds to stress, strain, and unaccustomed sensations.

You and Your Physician

The medical profession is often subjected to criticism—and sometimes rightfully so—for certain shortcomings. Yet, most of us in our individual association with our physician find far more to admire than to censure. We recognize that he, having made a study of human behavior, knows a great deal about "what makes us tick." We recognize, too, that his profession has made an enormous contribution to the welfare of humanity.

There are other, if lesser, reasons for the esteem in which we hold the physician. We admire him for the responsibility he carries, the trusts he will not betray, and the discipline he enforces upon himself, often at the risk of his own health and frequently at the expense of his family life.

What are the rewards of a medical career? Perhaps the one that the physician values most is the knowledge that his neighbors look upon him as an indispensable member of the community. He values, too, the privilege of intimate association with all kinds of people, the satisfaction of bringing new lives into the world, restoring the sick and injured to health, preventing diseases and accidents, and easing pain in hopeless illnesses. Such enduring rewards are known only to the physician dedicated and devoted to medicine, both as a science and an art.

Nutrition and a State Medical Society

The ultimate goal of all research and clinical investigation in nutrition is the improvement of the health of the people. To this end several progressive state medical societies have established programs which vary in scope and objectives but which have as their fundamental philosophy the "practical application" of our newer knowledge of nutrition.

One of the leading examples of such a progressive viewpoint may be found in the report of the Commission on Nutrition of the Medical Society of Pennsylvania *(Pennsylvania M. J.* 60:1113, 1957). The Commission's report may well be studied by appropriate commissions of other state medical societies for it represents an admirable example of what can and should be done.

In brief, the Commission has had two objectives: (1) The stimulation of interest in clinical nutrition at state and county levels; and (2) the dissemination of factual information on nutrition to both practicing physicians and the laity.

Among the numerous accomplishments of the Committee are the following:

A number of editorials on various aspects of applied nutrition appeared in the state medical journal.

Three exhibits were presented at the annual sessions of the state medical society. These dealt with obesity, electrolytes, and salt-free diets.

A Coordinating Committee was established with representatives from city, state, and health medical societies, dental societies, state nurse organizations, etc. This Committee undertook a survey of the extent of nutritional training in hospitals. It was learned, not unexpectedly, that most hospitals offer little or no training in nutrition to interns and residents. The great majority of institutions, however, expressed a desire for a manual on standard therapeutic diets for

both reference and teaching purposes. As a result, therefore, the Coordinating Committee prepared a manual of standard therapeutic diets which apparently has met with considerable enthusiasm state and nationwide. Copies of the manual have been distributed free of charge to senior medical students of the six medical schools in Pennsylvania.

As part of an education program, the State Nutrition Commission organized various symposia. These were held at State Medical Society meetings, and in cooperation with the National Vitamin Foundation, and with the Philadelphia County Medical Society.

The Commission has also urged the establishment of nutrition clinics throughout the state and supported a pioneering nutrition clinic now in operation at the Philadelphia General Hospital.

In cooperation with the Dietetic Association, a program will be prepared for the laity.

The Commission cooperated with the Pennsylvania Heart Association in the preparation of a manual on salt-free diets for free distribution.

The Commission also prepared a program of vitamin and other nutritional supplementation for patients under state public assistance. This doubtless will save the taxpayer a considerable amount of money.

The writer has had an opportunity to see a proposal for the establishment of a Division of Nutrition within the Department of Public Health of the City of Philadelphia. Because it is felt that this may be of interest to some of our readers, the following is a brief summary of this proposal.

The purposes of such a Division of Nutrition within a City Department of Public Health are (1) the promotion of better public health through research and special clinics; (2) the prevention of diseases arising from public ignorance of this field; and (3) direct participation in programs both in prenatal clinics, maternal nutrition studies, and school nutrition projects; and finally (4) rehabilitation of those who are in medical need from the public health standpoint, such as obesity, nutritional anemia, diabetes, etc.

The recommended organization would be located in a large city hospital. The staff would consist of a director, three physicians, two dietitians, a psychiatrist, a laboratory technician, two social workers, and a clerk. A number of laboratory studies would be performed including, in addition to routine determinations, analyses of vitamin C, urinary thiamine, urinary riboflavin, and electrolytes.

The Nutrition Division could plan courses for members of the Department of Public Health, such as public health nurses, dietitians employed by other state and city agencies, general physicians, and school physicians. Furthermore, this division could furnish consultants to other departments within and outside the Department of Health, would engage in the nutrition and education in medical schools and hospitals, help improve dietary practices in various state institutions, and advise various industrial hygiene divisions.

It is clear that a great deal may be offered by city- and state-organized medicine in improving the role of nutrition as a medical science and as an adjunct toward the health of the public.

A special commendation should be made to the Commission on Nutrition, State of Pennsylvania, under the chairmanship of Dr. Michael G. Wohl, whose members include Drs. T. E. Machella, R. E. Olson, H. N. Seiple, J. N. Seitchik, P. L. Shallenberger, Paul C. Shoemaker, J. M. Strang, and C. W. Wirts, Jr.

It is hoped that through these means some of the progress in clinical nutrition can be brought to a more practical application to the health of the people. —*S. O. Waife, M.D., American Journal of Clinical Nutrition, July-August 1958.*

Doctor Martin Heads Board of Health

Dr. Geoffrey M. Martin, former director of maternal and child health for the Kansas State Board of Health, Topeka, became executive secretary of the Board last month, succeeding Dr. Thomas R. Hood, who went to New York to become associate director of the American Public Health Association.

Dr. Martin, who received his medical degree from Tulane University, also holds a master's degree in public health from Harvard. He served in the Navy during World War II, after which he became assistant in pediatrics at Johns Hopkins Medical School. His first work in Kansas was in Cowley County.

Dr. Patricia Schloesser, who has been a part-time pediatric consultant with the Board since 1953, has been appointed director of the division of maternal and child health.

Pediatric Society Meeting

The annual meeting of the Kansas State Pediatric Society was held at Emporia on Saturday, September 6. A scientific program was presented, and officers for the coming year were elected at a business session. Those chosen are: president, Dr. Hilbert P. Jubelt, Manhattan; vice-president, Dr. Thomas C. Hurst, Wichita; secretary-treasurer, Dr. William H. Crouch, Jr., Topeka.

Clinicopathological Conference

Acute Renal Failure, Convulsions, Hematemesis, Hypertension and Vividialysis

Case Presentation

A 63-year-old white housewife was admitted to KUMC for the first time on February 1, 1957 complaining of oliguria for two days.

Five days before her admission to this hospital she slipped and fell, sustaining an intertrochanteric fracture of the left hip for which she was hospitalized elsewhere. Two days before admission here her hemoglobin was 68 per cent, and at that time she was given one unit of whole blood. A Smith-Petersen nail was inserted under spinal anesthesia, and her postoperative condition was considered good. At 2:30 p.m. of the same day, however, she complained of pain in the back and neck, and at 6:00 p.m. she became unconscious. Her blood pressure was not obtainable, and her extremities were cold. She was given dextran and levarterenol (Levophed) in 400 ml. of saline. Her blood pressure came up to 120/70, and she regained consciousness. Following that she was given 500 ml. of whole blood, 1000 ml. of dextran and levarterenol in saline, and 1000 ml. of 5 per cent dextrose in water, and her blood pressure rose to 162/90. She received a total of 3700 ml. of fluid following surgery, and during the next 48 hours her urinary output was 163 ml.

On February 1, 1957, she was brought to this hospital by air ambulance, arriving at 8:00 p.m. She had been told that she had low grade hypertension, but she denied having been ill. She apparently had had no other significant previous illness. Her mother died of cancer of the uterus; her father died of pneumonia.

The patient was a well developed white woman, conscious and cooperative, with a uriniferous odor to her breath. The head, eyes, ears, nose, throat, and skin were negative. The neck was supple, no thyroid was palpable, and there was no adenopathy. Her admission blood pressure was 170/90; pulse rate, 84, with a normal sinus rhythm. The PMI was in the fifth intercostal space to the left of the midclavicular line. Fine moist rales were heard throughout both lung fields. Bowel sounds were present, and no abdominal organs were palpable. There were bilaterally equal grade III deep tendon reflexes. The ocular fundi revealed grade I to II arteriosclerotic changes. There was a cast on her left leg.

The specific gravity of the admission urine specimen was 1.010 with an acid reaction, a trace of albumin, and no sugar. There were 15 to 20 pus cells, 40 to 50 red blood cells, and occasional granular casts per high power field. The white count was 16,750 with 93 per cent polymorphonuclears, 4 per cent lymphocytes, and 5 per cent monocytes. The hematocrit was 50 ml., and the hemoglobin was 15.6 gm. per cent. The serum sodium was 136 mEq/L; potassium, 4.6 mEq; chloride, 99 mEq; calcium, 3.9 mEq.

Shortly after admission she had a grand mal seizure which was controlled by intravenous magnesium sulfate. On February 2 the blood urea nitrogen was 80 mg. per cent; creatinine, 5.8 mg. per cent; fasting blood sugar, 91 mg. per cent; carbon dioxide, 16.4 mEq/L; serum sodium, 136 mEq; potassium, 4.7 mEq; calcium, 3.6 mEq; phosphorus, 3.4 mEq. The sedimentation rate was 16 mm. in 60 minutes.

Her fluid intake was restricted to 20 ml. on February 2, and on February 3 she took no fluids. On February 4 her blood pressure dropped from 170/90 to 150/70 and remained at this level until February 6, gradually rising again to 170/70 on February 7.

She was started on a protein- and potassium-free, high caloric diet consisting of carbohydrates and fat. Calories were supplied by butterballs and sugar candy, but she became nauseated and her caloric intake was poor.

The blood urea nitrogen gradually rose to 131 mg. per cent on February 3; 161 and 206 mg. per cent on February 5, and 225 mg. per cent on February 7. The creatinine rose steadily to 14.5 mg. per cent on February 7. The carbon dioxide combining power decreased daily, and on February 8 it was 10.1 mEq. The serum sodium was 136 mEq initially and rose to 143 mEq; the potassium rose from an initial level of 4.6 mEq to 5 mEq on February 3; 4 to 5.6 mEq, February 4; 6.1 mEq, February 5; 6.4 mEq, February 6; and 6.7 mEq on February 7. The serum chloride level was 101 mEq on February 4 and 95 mEq on February 8. On February 1 the serum calcium was 3.9 mEq, and on February 8 was 3.2 mEq. The inorganic

Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center, as well as by the third and fourth year classes of students.

phosphorus was 6.9 mEq on February 6. Electrocardiograms were taken daily.

Her urinary output on February 2 was 135 ml. and slowly increased with daily volumes of 140, 195, 170, 175, 160, 235, 325, 460, and 350 ml. Vividialysis with an artificial kidney was performed on February 7. The blood urea nitrogen was reduced from 225 mg. per cent to 161 mg. per cent, and the creatinine from 14.5 mg. per cent to 12.0 mg. per cent. During the vividialysis the blood pressure rose, and the blood flow through the artificial kidney was not satisfactory. Following the vividialysis the blood urea nitrogen rose steadily and the carbon dioxide combining power decreased. She became nauseated and vomited on February 6; this ceased following the first vividialysis, but recurred two days later. Her fluid intake ranged from 400 ml. to 1000 ml. in 24 hours. Her blood pressure was 170/70 on February 10 and 180/70 on February 11.

At 2:00 p.m. on February 12 she complained for the first time that she was unable to focus her eyes. At 4:00 p.m. she had slight twitching of the face and extremities, and she was given intravenous amobarbital sodium. Her convulsive movements continued, and at 7:00 p.m. she again received 200 mg. of amobarbital sodium intravenously. At that time she appeared to be slightly confused, and her convulsive movements continued despite medication.

She was placed on the artificial kidney for the second time at 11:00 p.m., and at 1:00 a.m., February 13, she vomited 50 ml. of bright red fluid. During the second vividialysis her hypertension increased. Small amounts of blood were withdrawn from time to time and antihypertensive medications were given. Her diastolic blood pressure rose from 75 to 90 mm. Hg. During the fifth hour of vividialysis the systolic pressure was 225 and the diastolic pressure was 90, dropping precipitously following the intravenous administration of 0.5 mg. of pentolinium. At that time her respiration was irregular and slow; her pupils were dilated and fixed; and she was unresponsive. In the sixth hour of dialysis her blood pressure remained 150/80 for 30 minutes and then began to drop. Artificial respiration with the positive pressure respirator was begun, and she was given levarterenol in saline. At 6:50 a.m. on February 13 her heart ceased to beat, and she was pronounced dead. Her temperature had remained normal until the final day when it rose to 104.8 degrees, rectally, and gradually fell to 101 degrees just before death.

Dr. Mahlon Delp (moderator): Are there any questions?

John Chapman (fourth year medical student)*: What were the chest findings on her last hospital day?

Dr. Wu-Hao Tu (resident in medicine): A few rales were heard.

Gordon Cowles (student): Did she have a previous history of kidney disease?

Dr. Tu: No, but she had a history of hypertension.

Alfred Cheng (student): What was the serum potassium level during her hospitalization?

Dr. Tu: On February 2 the potassium was 4.7 mEq; February 3, 5 mEq; February 4, 5.6 mEq; February 5, 6.1 mEq; February 6, 6.4 mEq; February 7, 6.7 mEq; February 8, 6.2 mEq; February 9, 6.1 mEq; February 10, 6.0 mEq; February 11, 6.1 mEq; and on February 12 it was 4.4 mEq.

Milton Claassen (student): Did she ever receive digitalis?

Dr. Delp: On her last day 0.6 mg. of digitoxin was ordered, but I am not sure that it was given at that time. Dr. Allen, do you know anything about this?

Dr. Max Allen (internist): I believe it was given.

Craig Clark (student): Did she have a stiff neck on her last hospital day?

Dr. Tu: No, she did not.

Billy Cooney (student): Did she ever have dark urine?

Dr. Tu: It was not grossly dark.

Mr. Cheng: Did she vomit anything other than the 50 ml. of bright red fluid?

Dr. Tu: No, she did not.

Donald Coldsmith (student): Did she ever have melena?

Dr. Delp: It was never reported.

Dr. Thomas J. Rankin (internist): Did she have severe pain in the neck and back on the postoperative day?

Dr. John F. Christianson (internist): She had some mild back pain, but I do not believe it was terribly severe.

Mr. Cowles: Was the first unit of blood given before or after the operation?

Dr. Tu: It was given in the morning before the operation.

Mr. Clark: What was the platelet count during the second dialysis?

Dr. Tu: It was 174,000.

Mr. Coldsmith: What was the total amount of heparin given, and what was the nature of the electrolytes in the artificial kidney?

Dr. Tu: Initially she received 145 mg. of heparin; the second time she received 148 mg. Before that the serum sodium was 123 mEq, and the chloride was 110 mEq.

Dr. Charles Poser (neurologist): Was she fully alert when she was admitted?

Dr. Tu: She was fully alert on admission, but shortly afterward she had grand mal convulsions.

*Though a medical student at the time of this conference in January, 1958, he, like the others referred to as students, received the M.D. degree in June, 1958.

Dr. Delp: Now, if there are no further questions, we shall see the electrocardiograms.

Mr. Coldsmith: The electrocardiogram taken on the day of admission shows a regular sinus rhythm with a rate of approximately 80 per minute. I see no abnormalities, and I interpret this as a normal tracing.

The next electrocardiogram, taken on February 7, approximately one week after admission, shows peaking of the T wave but no other abnormalities. I interpret this as a normal tracing with the exception of the peaked T waves.

A tracing taken on February 11 again shows a regular sinus rhythm with a rate of approximately 80 per minute. There is some indication of peaking of the T waves, but this is not so marked as in the previous tracing, and I interpret this as an essentially normal electrocardiogram with the exception of the peaking of the T waves.

A tracing taken on the day of death (Figure 1) shows evidence of sinus rhythm with ectopic beats, the rate varying from 100 to 120 per minute. There are biphasic waves and some indication of flattening of the P waves and depression of S-T segments which I believe may be attributed to digitalis intoxication or hyperkalemia.

Dr. Delp: Dr. Crockett, do you have any comment?

Dr. James E. Crockett (cardiologist): I cannot be sure from the last tracing whether P waves are present. Potassium intoxication may produce auricular standstill as well as the other changes which are apparent here.

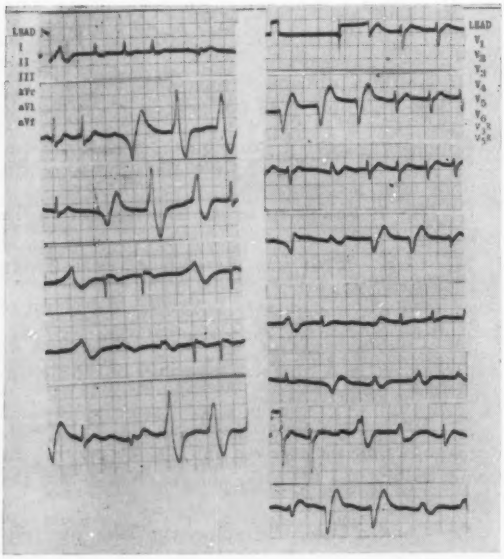


Figure 1. Electrocardiogram taken on the day of death.

Dr. Delp: May we have the x-rays, please?

Mr. Clark: Two x-rays were taken on the day of admission. No bony abnormalities are seen in the chest film, the trachea is in the midline, and the costophrenic angles are clear. There is a slight increase in the mediastinum and in the bronchovascular markings. The heart is at the upper limits of normal. The film of the pelvis and the proximal part of the femur shows a bony abnormality with the fracture line and the Smith-Petersen nail in place. A bony fragment is also seen which is probably the lesser trochanter. There is gas in the bowel.

Dr. Delp: May we have your discussion now, Mr. Cooney?

Differential Diagnosis

Mr. Cooney: I shall base my differential diagnosis on the causes of oliguria and acute renal failure. First, I shall rule out obstructive uropathy as a possibility because there were no historical or clinical findings to support this diagnosis. Acute nephritis can be ruled out because of the clinical course and the absence of the typical laboratory findings. Nephrotoxins can also be dismissed because there was no history of exposure to them.

It is difficult to rule out bilateral renal artery thrombosis, but I believe our patient had acute tubular necrosis, secondary to postoperative shock and prolonged renal ischemia. It becomes difficult to determine whether she had a transfusion reaction from the blood that she received before surgery or whether she had a vascular collapse secondary to a surgical trauma. There is said to be no definite etiology in 45 per cent of patients with acute tubular necrosis. Two days before her admission to this hospital she underwent surgical repair of her intertrochanteric fracture on the left hip, and four hours later she went into shock. Routine treatment for shock was given, and she was given a total of 3700 ml. of fluid containing at least 1000 ml. of blood. During the next 48 hours she was oliguric with an output of only 163 ml., and she was transferred to this hospital for further evaluation.

On admission she had a uriniferous odor to her breath which, with the elevated blood urea nitrogen and creatinine, indicated advancing azotemia. The urinalysis showed a specific gravity of only 1.010 which, when associated with oliguria, is highly suggestive of renal failure, and I believe this can be attributed to her hypervolemic state. Shortly after admission she suffered a grand mal seizure, probably attributable to cerebral edema. She was started on a protein-free, high caloric diet consisting of carbohydrates and fat, but despite all measures the blood urea nitrogen, creatinine and metabolic acidosis increased. During that time there was a gradual increase in urine volume which might have been indicative of regener-

ation of some renal tubules. On the seventh hospital day she was dialyzed, and this resulted in a sudden rise of blood pressure and a reduction of the blood urea nitrogen. After dialysis, however, there was a progression of her azotemia and acidosis. On the eighth day she developed vomiting, probably from uremic gastritis. On the 12th day neurologic findings appeared which I believe were again due to cerebral edema. Marked twitchings developed, but these were fully controlled with amobarbital sodium.

She was again dialyzed, and her blood pressure rose much higher than before, being controlled with difficulty. She began to vomit bright red fluid. Her breathing became irregular, and artificial respiration was started. She became comatose, and soon afterward her heart stopped beating.

In summary, several things must be considered as the cause of our patient's death. First, there was the bleeding tendency secondary to her dialysis. Patients who are dialyzed often develop a bleeding tendency, and our patient was known to have vomited 1500 ml. of bright red blood. We do not know how much more was left in the gastrointestinal tract. Secondly, pulmonary edema is a common cause of death in patients who have hypervolemia and oliguria.

The electrocardiogram showed evidence of potassium intoxication. As the potassium increases the heart tends to contract irregularly often resulting in ventricular fibrillation.

It is difficult to say whether she died of a cerebral vascular accident due to a bleeding tendency or whether she died of potassium intoxication.

Clinical Discussion

Dr. Delp: Thank you, Mr. Cooney. This case was selected because I wanted some comments and discussion on the etiology of acute renal failure and what I choose to call a conservative versus a more vigorous form of treatment in patients with at least a clinical picture similar to this. Also, because the artificial kidney was used, I wanted comments in regard to the uses of this instrument and other pertinent items having to do with the use of any gadget in the treatment of patients. Mr. Chapman, can you give me one reason for our patient's renal failure?

Mr. Chapman: I believe it was renal ischemia secondary to shock.

Dr. Delp: Mr. Claassen?

Mr. Claassen: I agree with that.

Dr. Delp: Mr. Cheng?

Mr. Cheng: It could have been a transfusion reaction.

Dr. Delp: Mr. Cowles?

Mr. Cowles: I believe it was most likely secondary to the surgical trauma.

Dr. Delp: Then you believe this was a delayed phenomenon occurring four or five hours after the patient left the operating room?

Mr. Cowles: I do not know exactly when she left the operating room, but it occurred afterward.

Dr. Delp: Mr. Clark?

Mr. Clark: I agree that it was due to the surgical procedure.

Dr. Delp: Then you believe it was a delayed shock occurring four hours after surgery?

Mr. Clark: Usually the shock occurs in 20 minutes after a spinal anesthesia such as our patient had.

Dr. Delp: Mr. Coldsmith?

Mr. Coldsmith: I believe it was transfusion reaction and surgical shock.

Dr. Delp: Mr. Chapman, what do you believe was the cause of death?

Mr. Chapman: Potassium intoxication.

Dr. Delp: Mr. Cowles?

Mr. Cowles: I believe it was a low serum potassium, and I believe that digitalis intoxication should be ruled out.

Dr. Delp: Do you believe that digitalis intoxication caused our patient's death, Mr. Cheng?

Mr. Cheng: A high magnesium level may cause a patient to become comatose.

Dr. Delp: Will you tell us about magnesium intoxication and the part it might have played in this case?

Mr. Cheng: At times, in a patient with oliguria, there is retention of magnesium, but because there is no laboratory evidence to support this, I am merely offering it as another possibility.

Dr. Delp: Do you believe it might have been just as valuable as a determination of the potassium levels?

Mr. Cheng: The magnesium level rises out of proportion in some patients with oliguria.

Dr. Delp: Are you familiar with the electrocardiographic picture in this situation?

Mr. Cheng: I believe there is left bundle branch block.

Dr. Delp: Mr. Cowles, what do you believe was the cause of our patient's death?

Mr. Cowles: Digitalis intoxication.

Dr. Delp: Mr. Clark?

Mr. Clark: An intracerebral vascular phenomenon and hemorrhage which could have been precipitated by dialysis.

Dr. Delp: Mr. Coldsmith?

Mr. Coldsmith: I believe it was caused by digitalis intoxication and cardiac arrest.

Dr. Delp: Mr. Cooney, what is your opinion?

Mr. Cooney: I believe the cause was digitalis intoxication with a lowered potassium level due to dialysis.

Dr. Delp: There are four of you who believe she

died of digitalis intoxication. Mr. Chapman, how do you believe the pathologist will describe the kidneys?

Mr. Chapman: The kidneys should appear large and edematous, and there should be evidence of regeneration in the tubules, and there should be spotty necrosis throughout all of the tissue. There may be hyaline, protein, and hemoglobin casts.

Dr. Delp: Do you believe the tubules will be obstructed?

Mr. Chapman: I do not say that this is an etiology of acute tubular necrosis, but I believe the tubules will contain casts.

Dr. Delp: What is your opinion about conservative versus vigorous treatment of renal failure, Mr. Cooney?

Mr. Cooney: It has been stated that about 80 per cent of patients with this syndrome will recover with conservative treatment.

Dr. Delp: Mr. Coldsmith?

Mr. Coldsmith: I believe that conservative treatment may improve the patient's condition in 80 per cent of the cases. On the other hand, some people see three indications for the use of the artificial kidney: elevated potassium, progressive azotemia, and convulsions. Our patient had all three of these. I believe that on the first occasion she received some benefit from the use of the kidney as evidenced by the lowering of the blood urea nitrogen. She did have complications when it was used the second time, and I do not believe that she benefited from it.

Dr. Delp: Mr. Clark?

Mr. Clark: I believe that conservative treatment should have been tried.

Dr. Delp: Mr. Cowles?

Mr. Cowles: I agree that conservative treatment would probably be better, but in our particular case it is impossible to say whether or not she benefited or was harmed by the artificial kidney.

Dr. Delp: Mr. Cheng?

Mr. Cheng: I would have treated this patient conservatively.

Dr. Delp: Mr. Claassen?

Mr. Claassen: I believe conservative treatment would have been better.

Dr. Delp: Mr. Chapman?

Mr. Chapman: Conservative treatment should be followed until the three indications mentioned earlier are found.

Dr. Delp: Dr. Bolinger, may we have your comments, please?

Dr. Robert Bolinger (internist): As far as the original etiology is concerned I have nothing to add, but I am particularly interested in our patient's response to the dialysis. The possibility has been mentioned of potassium deficiency and the effect of the digitalis together producing a cardiac complication. Several factors are against this. In the first place, during the terminal event she first showed changes in consciousness, and then for some time her blood pressure was not unobtainable, but was 150/80. I believe that this is a factor against a primary cardiac dysrhythmia. Secondly, I am not impressed with the efficiency of the artificial kidney in removing potassium from the body so rapidly. I doubt that the hypermagnesium was contributory since the dialysis fluid contains magnesium in physiological amounts, and there should be a gradient, if there were a hypermagnesium in the first place, which would eliminate magnesium from the body.

Other things that must be considered when the patient's condition becomes worse during dialysis include dynamic changes which may occur because of too much fluid going into or out of the patient's blood. I doubt that that was a factor in our case. Transfusion reactions must be considered and inaccuracies in the osmolarity of the bath fluid producing either hypo-osmolarity or hyperosmolarity. In our patient's case I believe that this should be considered because of her mounting blood pressure and the change in consciousness, all consistent with cerebral edema characteristic in such conditions. Finally, we must consider the hypertensive reaction which goes along with this. I believe, in the final analysis, that our patient's death was due to intracranial complications, probably an intracranial hemorrhage from the rise of blood pressure and remembering the heparin which had to be given.

Dr. Delp: Thank you, Dr. Bolinger. May we have your opinion, Dr. Poser?

Dr. Charles Poser (neurologist): I saw this patient a few hours before her death, and at that time she was deeply comatose. Her pupils were dilated and fixed. I was impressed with the fact that before she became comatose she had complained of some difficulty with her vision, and perhaps I erroneously interpreted this as diplopia. There is little doubt that severe metabolic disturbances such as she had accounted for much of her difficulty. She probably had a great deal of cerebral edema, and I assume that she was atherosclerotic. With the artificial kidney her brain was subjected to sudden and wide changes in hemodynamics, her blood pressure rising and falling rather rapidly. In addition to this, there were changes in the potassium level and in her calcium and phosphorus relationship.

In regard to the terminal event I am not sure whether she died of hemorrhage or thrombosis. My belief is that, rather than massive hemorrhage, she had multiple petechiae especially in the brain stem. She may have herniated, and, as a result of these

hemorrhages and softening of the brain stem, died. All of this was the result of the wide range of blood flow through her vascular system. I have nothing to add about the primary etiology, but there might have been the possibility of a fat embolism. However, it is most unusual in hip fractures, and the course is certainly not that which is found in this disease.

Dr. Delp: Thank you. Dr. Allen, will you give us your opinion, please?

Dr. Allen: Dr. Poser's mention of fat embolism deserves some consideration. Classically, of course, it does occur after the fracture of long bones and, after an interval free of symptoms, the patient first develops respiratory deficiencies, cyanosis, and periods of shock. I believe this factor had to be considered on the operative day as a cause of her cyanosis and shock. Following this, one sees the classic cerebral symptoms of fatty embolism of the brain. So the time relationships appear good for fat embolism, but the fact that the fracture was in the neck and not down in the shaft of the femur is against this. There were no petechiae of the skin, classically seen in the lateral axilla, and which one might expect to see in fat embolism. It is my belief that the initial shock was associated with the surgery.

The final episode occurred in a dramatic fashion. Her blood pressure rose gradually for a while, then rather precipitously, and it was at that time that she vomited the bloody fluid. Shortly afterward she became comatose. Her pupils rapidly became dilated and fixed. This appeared to me to be an apoplexy, and my opinion at that time was that she had an intracerebral hemorrhage. I am uncertain whether this occurred in an area of a previous lesion of fatty embolization with brain softening around it, as a result of some other lesion there, or simply as a result of anticoagulation and a sudden rise in blood pressure. Her condition deteriorated rapidly, and in spite of the artificial respiration she was poorly oxygenated. The last electrocardiogram, taken only a few minutes before death, seemed to agree with a terminal myocardial hypoxia.

In general, I am conservative about using gadgets or machines in the treatment of patients. As has been pointed out, two or three things were occurring here that disturbed us. Perhaps there was some other cerebral reason for the diplopia and the convulsions. Perhaps simple cerebral edema was the only cause. But these things were getting worse, and it became evident that the patient was going to be in real trouble and in a convulsive state if we could not correct it. I do not believe that she was ever in any real danger from her hyperpotassemia, and I do not consider hyperpotassemia to be the chief indication for dialysis. The high blood urea nitrogen and the convulsive state were the main reasons for trying another procedure.

Dr. Delp: Dr. Allen, had the patient stopped breathing before artificial respiration was begun?

Dr. Allen: Yes.

Dr. Delp: Do you believe that she was breathing or could have been breathing on her own for that 16-hour period?

Dr. Allen: I am certain that she could not.

Dr. Delp: Dr. Rasmussen will now give the pathologist's report.

Pathological Report

Dr. Peter Rasmussen (pathologist): The changes found at gross autopsy could be arranged into four groups. First were those due to old age, including atrophy of the ovaries, a moderate degree of osteoporosis, and the internally fixed fracture through the trochanter of the left femur.

The second major category were changes in the kidneys. They were not enlarged, but there was a distinct cortico-medullary junction which made the gross diagnosis of lower nephron nephrosis tenable.

The third category was the evidence of heart failure. This apparently had been progressing for some time, as there was a distinct "nutmeg" character of the liver, bilateral pleural effusion, and minimal edema of the legs.

In the fourth category were manifestations of hemorrhage in the brain and heart. The brain weighed

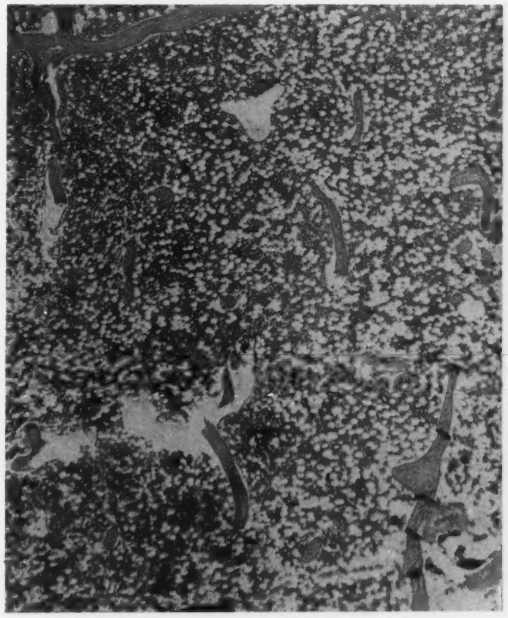


Figure 2. Photomicrograph of a lumbar vertebra illustrating osteoporosis. Note the thin bony trabeculae. Hematoxylin and eosin stain. 96X.

1,480 grams and contained three large areas of recent hemorrhage. One, in the white matter of the left hemisphere extending into the occipital lobe, measured 6 x 5 x 4 cm. The second, measuring approximately 2 cm. in diameter, was on the opposite side at the same level as the first. The third, measuring approximately 3 x 2 x 2 cm., was at the level of the mamillary bodies in the white matter of the right hemisphere. Finally, there was a large area of subendocardial hemorrhage along the bundle of His in the left ventricle.

Microscopically, the ovaries were largely replaced by dense pink collagen except for a small rim of cortical tissue. Consequent to this was moderate generalized osteoporosis as seen in the lumbar vertebra, and documented by thin bony compacta and the paucity of bony trabeculae in the vertebral body (Figure 2).

While there was no gross callus formation in the area of fracture, there was beginning callus and organization of the hematoma and the laying down of osteoid along small osteoporotic bony trabeculae. Osteoblasts were arrayed in "soldier fashion" along the newly formed osteoid (Figure 3). In addition, there were necrotic bone fragments being removed by osteoclasts.

The kidney demonstrated lower nephron nephrosis. Chronic phlebitis could be seen around thin-walled arcuate veins, and this change was mainly present at the cortico-medullary junction. In addition to numerous hemoglobin casts, homogenous protein casts were evident mainly in the distal convoluted tubules. Some of these had ruptured into the interstitial tissue with surrounding chronic inflammatory cells, the so-called cast granuloma (Figure 4). These may herniate into an adjacent vein resulting in what has been called a tubulo-venous fistula. Finally, at this stage of development of hemoglobinuric nephrosis there were mitoses in tubular cells indicating regeneration.

The parathyroid glands revealed a diminution of parenchymal fat, as well as a tendency toward acinar formation in glandular cells. In addition, numerous mitotic figures were evident. In my opinion, this picture represented a reaction to the uremia.

Throughout both right and left ventricles there were several areas of subendocardial micro-infarction. Hemosiderin-laden macrophages were scattered in the loose granulation-type tissue, and I believe that the presence of these macrophages indicated that the infarcts occurred at the time that the patient had her

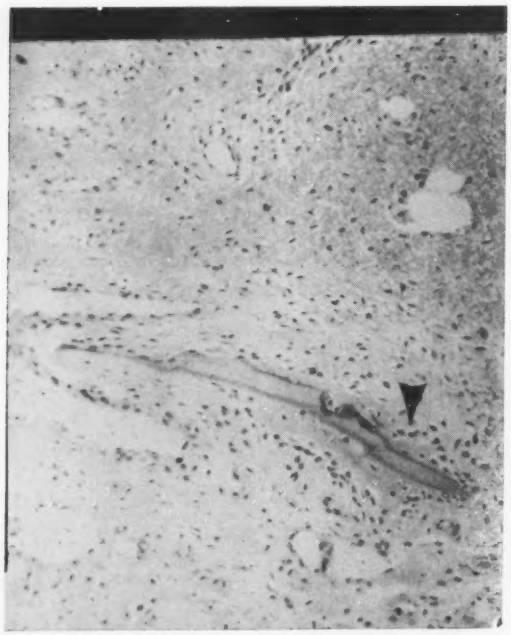


Figure 3. Early callus formation is evident in the hip fracture. An organizing hematoma is seen in the top one-half of the field. A bony trabecula has osteoid being laid down upon it. The arrow indicates a row of osteoblasts. Hematoxylin and eosin stain. 157X.

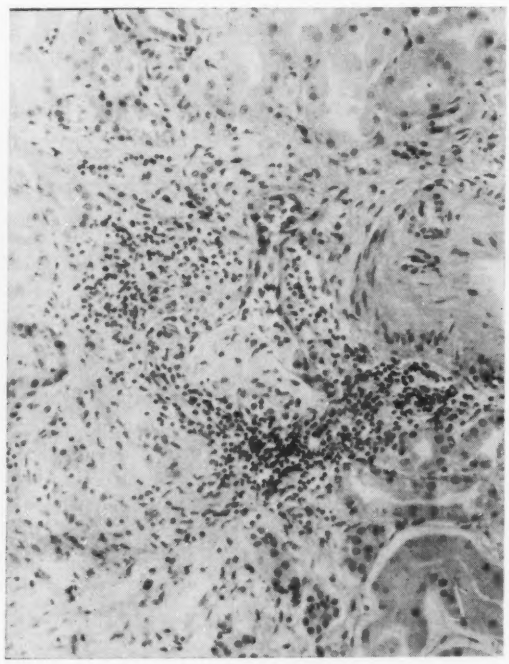


Figure 4. A cast granuloma of the kidney showing protemaceous material rupturing through a distal convoluted tubule and exciting the chronic inflammatory response surrounding it. Hematoxylin and eosin stain. 175X.

episode of hypotension just after her original hip nailing, approximately three weeks before death.

Cerebral edema was present throughout the brain, substantiated by the increase in weight and seen microscopically as perineuronal vacuolization. Other areas of the brain adjacent to the regions of gross hemorrhage showed only recent extravasations of blood in the form of perivascular, satellite hemorrhages. Small petechial hemorrhages could also be found in the brain stem, mainly in the sections of the pons. Multiple sections through the brain failed to reveal any micro-infarcts which one might expect subsequent to fat embolization after a fracture of this duration; indeed, fractures through the femoral neck area are not prone to cause fatty emboli.

Finally, in the liver there was chronic passive congestion with hemosiderin in Kupfer cells in the central areas as well as necrosis of hepatic cells. A slight increase of central fibrous connective tissue accompanied this.

Pathogenetically the problem arises as to the cause of the grand mal seizure which occurred approximately 11 days before death. We are left with the speculation that overhydration immediately following her hypotensive state may have contributed to cerebral edema, which, in turn, may have been the precipitating factor for the seizure. Finally, it seems that there is difficulty in establishing the precipitating factor of the cerebral hemorrhages which caused her final episode. My first thought was that dialysis by the artificial kidney might have been involved, since an anticoagulant is used in the procedure. That vividialysis was probably not of prime importance is revealed in the history because visual difficulty as well as the final episodes of convulsions occurred before the second dialysis. Also, the neurologic manifestations occurred three days after the first vividialysis, thus making this event seem unlikely as a precipitating cause. It seems that some bleeding took place before her second dialysis, but perhaps the second dialysis, because of the use of heparin, may have enhanced the cerebral hemorrhage. In searching for a primary cause of the hemorrhage we are left with uremia or cerebral edema as predisposing factors.

Dr. Delp: Dr. Bolinger, are you willing to accept the final episode as being a complication of dialysis?

Dr. Bolinger: It is a good possibility, but the patient's course was directed that way, and it just seemed to hasten it.

Dr. Delp: Are such situations a complication of dialysis, or are there other complications we should keep in mind when using this procedure?

Dr. Bolinger: I do not believe there are any complications particularly applicable to our patient. There is the possibility of over-hydration, as Dr. Rasmussen mentioned. This could be accounted for if the bath were hypotonic.

Dr. Delp: Thank you. I believe the principal features of this problem have been well illustrated.

Summary

Dr. Delp: Acute tubular necrosis so prominently cited in the descriptions of complications occurring in wartime casualties can and does feature events of almost routine care of an illness which in itself seems minor. The prompt recognition and treatment of shock is an important factor in prevention.

Once the state is recognized the desire to force diuresis by excessive fluid input must be controlled. A decision to use vividialysis should then be made only after a careful consideration of all of the factors operating in the patient. Here previously existing hypertension, the age of the patient, pre-existing vascular disease, and cerebrovascular insufficiency suggested that vividialysis might subject the patient to greater danger than more conservative measures.

Pathological Anatomical Diagnosis

Recent massive hemorrhages into the cerebrum, brain stem, subarachnoid space, and ventricles.

Edema of the brain, weight 1480 gm.

Small infarcts of the myocardium consistent with two to three weeks duration.

Recent hemorrhage in the endocardium.

Bilateral pleural effusion, acute passive congestion of the liver with slight chronic passive congestion, and edema of the lower extremities.

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Harold G. Ingham Dies

Mr. Harold G. Ingham, former executive director of the extension program in medicine at the University of Kansas Medical Center, died there on August 21 after suffering a heart attack. He had been employed by the university since 1915, serving first as director of the correspondence study bureau in Lawrence and moving to the Kansas City position in 1945. Since retiring from the university position, he had been executive secretary of the Kansas Adult Education Association.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

When the Congress that is elected in November goes to work next January 7, it will have before it a half dozen important health-medical issues that the last Congress took some interest in but didn't resolve. They include hospitalization under social security, tax-deferment on annuities, loans and mortgage guarantees for hospitals and nursing homes, aid to medical schools, and amendment of Veterans Administration hospitalization procedures.

The issue of hospitalization under social security—the Forand bill principle—will come into the spotlight shortly after the new session starts. Under instructions from the House Ways and Means Committee, the Department of Health, Education, and Welfare will complete a study on the problems of financing hospital care for the aged before next February 1. Some study of medical costs may also be included.

Decision to move ahead with a study of medical care costs for the aged was reached by the committee at the same time it excluded the Forand idea from the social security bill enacted during the summer. HEW was told to pay particular attention to the possibility of increasing OASI taxes, and with the money purchasing health insurance (nonprofit or commercial) to take effect upon retirement or disability. This would differ from the Forand plan in that health care would be financed through insurance, and not paid for directly by the federal government.

The Keogh bill to allow doctors and other self-employed to defer income taxes on money put into retirement funds passed the House with little opposition but encountered difficulty in the Senate. It was defeated there in the closing days, and under unusual circumstances. Policy committees of both parties decided to oppose the bill as too costly, and the vote came in the course of a complicated legislative maneuver that could not be used as a test of whether individual senators favored or opposed the bill itself.

Keogh bill sponsors, however, are encouraged that 32 senators resisted official party instructions and stayed with the pension plan. They are confident that next year under more favorable legislative circumstances the measure will clear the Senate.

An effort was made late in the session to authorize grants to medical schools for building and equipping teaching as well as research facilities. The bill extending the research grants program also would have allowed use of the grants for "multi-purpose" structures (teaching and research) if emphasis were on research. However, for fear this change would hold up the simple extension bill, it was dropped off before the bill reached the House floor. Sponsors of aid to medical education will be back next year and campaign on this issue alone.

Legislation for U. S. guarantee of nursing home mortgages, strongly supported by the American Medical Association, fell by the wayside in the House during the closing hours of the session, after having cleared the Senate with no trouble whatever. This also will be pushed next year, and may have a better chance of passage because of the growing emphasis on need for solving the problems of the aged.

Far too late for passage, Chairman Olin Teague's House Veterans' Affairs Committee reported out a bill that would make a number of changes in VA hospitalization procedures, liberalizing some and tightening up on others. The bill also would require VA to open 5,000 beds over which Mr. Teague and VA Administrator Whittier have been squabbling for months, the latter maintaining that the beds aren't needed. That issue still is unresolved, inasmuch as the bill didn't pass.

Congress did roll out a sizable list of medical-health laws. It ordered the calling of a 1961 White House Conference on the Aging, gave Food and Drug Administration authority to enforce its pre-testing standards on foods to which chemicals and other substances have been added, authorized loans as well as grants under the Hill-Burton program, authorized grants for the country's schools of public health and for civil defense purposes, raised military and VA physicians' pay, and required labor and management health and welfare plans to make reports and open up their books for inspection by members.

American Medical Association was able to persuade the Department of Defense and the administration to retain the post of assistant secretary (health and medical) in the reorganization of the department. In legislation passed by Congress to bring about the reorganization, one of the assistant secretary posts would have been eliminated, and the medical assistant was marked for down-grading. However, Secretary McElroy eventually announced that the position would be continued.

Even before Congress adjourned, it was clear that trouble was in sight for Medicare because of inadequate appropriations and instructions from Congress not to exceed the appropriation. To keep within the limitation, if possible, Defense Department was channelling many thousands of service families to military facilities, and at the same time limiting the scope of care permitted in civilian facilities.

PHYSICIANS' ACTIVITIES

Dr. E. Grey Dimond, director of the Cardiovascular Laboratory at the University of Kansas Medical Center, will be one of the speakers at the annual conference of the Oklahoma City Clinical Society, October 27-29.

A feature story in the *Eureka Herald* recently paid tribute to **Dr. Fred D. Lose,** Madison, who is now in his 53rd year of medical practice.

Dr. E. Dale Peffley, formerly of Chetopa, is now practicing in Hoisington in association with **Dr. Robert Moore** and **Dr. Charles Davenport.** Dr. Peffley is a graduate of Oklahoma University School of Medicine.

Two physicians from the University of Kansas School of Medicine, **Dr. Mahlon H. Delp** and **Dr. Robert T. Manning,** were speakers at a meeting of the Mississippi Valley Medical Society in Chicago, September 24-26.

Dr. A. C. Armitage, Hutchinson, has closed his office because of poor health.

A physician new in the state, **Dr. William C. Strutz,** is now serving two hospitals as radiologist, the Atchison Hospital at Atchison and the Cushing Memorial Hospital in Leavenworth. Dr. Strutz is a graduate of Marquette University School of Medicine, and he has practiced in Milwaukee and Detroit, taking his radiological training in the latter city.

Dr. Orville S. Walters, formerly of McPherson, has been appointed director of health services, professor of hygiene, and lecturer in psychiatry at the University of Illinois. He is a graduate of the Menninger School of Psychiatry, Topeka.

The Sumner County Selective Service Board recently honored **Dr. Harry L. Cobean,** Wellington, for 15 years of gratuitous service as its medical advisor.

Dr. Harold Lawless, who has been in practice in Sacramento, California, since his graduation from the University of Colorado School of Medicine, has begun practice in Blue Rapids.

Two physicians who are graduates of the University of Kansas School of Medicine, **Dr. James L. Barber** and **Dr. Dale W. Anderson,** have begun practice in Augusta. Dr. Barber has just completed internship at Broadlawns Hospital, Des Moines, and Dr. Anderson had his internship at Good Samaritan Hospital, Phoenix.

Dr. Bennett Wong, fellow of the Menninger School of Psychiatry, Topeka, has been named acting clinical director of the Winfield State Hospital and Training School.

DEATH NOTICES

Henry Osmond Hardesty, M.D.

Dr. H. O. Hardesty, 88, an honorary member of the Northwest Kansas Medical Society since 1943, died at his home in Jennings on August 17. He had been in retirement because of poor health during the past five years, but had been in practice more than 50 years before he retired. He was graduated from Kansas Medical College in 1900 and spent his entire professional life in Jennings with the exception of a period during World War I when he served in Europe in the Army Medical Corps.

Paul A. Binter, M.D.

A Wichita physician, Dr. Paul A. Binter, 43, died at his home on August 26 after suffering a heart attack. He had practiced in Wichita since 1951. A graduate of the University of Kansas School of Medicine in 1942, Dr. Binter served in the Army Medical Corps for three years during World War II, principally in Europe. He later had residencies at the Veterans Administration hospitals in Wichita and Excelsior Springs and became a diplomate of the American Board of Internal Medicine. He was an active member of the Sedgwick County Medical Society.

The psychological effects of tuberculosis were discussed by **Dr. Roy A. Lawson, Jr.,** of the Tuberculosis Hospital at Chanute, at the September meeting of the Allen County Medical Assistants Society in Humboldt.

Dr. Charles C. Gullett has been appointed director of medical services for Trans World Airlines, Kansas City. He has been with T.W.A. since 1953.

"The Clinician as a Sabbatician" is the subject of a paper by **Dr. E. Grey Dimond,** of the University of Kansas Medical Center, published in the September issue of *Missouri Medicine.*

Dr. Franklin D. Murphy, chancellor of the University of Kansas, Lawrence, recently spent a month in Latin America inspecting institutions of higher education.

Dr. Francis R. Applegate, a 1955 graduate of the University of Kansas School of Medicine, is now practicing in St. Marys in association with **Dr. Fred E. Brown.**

The Nelson Clinic, Manhattan, announces that **Dr. Robert D. Olney** is now a member of its staff, specializing in surgery. A graduate of the University of Nebraska College of Medicine, he was a resident surgeon at the university's hospital in Omaha after he completed his internship there.

Dr. Jay H. Armstrong, Hutchinson, has resigned as county physician for Reno County to devote his time to private practice.

A newcomer in Great Bend who is practicing ophthalmology in association with **Dr. David T. Loy** and **Dr. Robert C. Polson** is **Dr. William F. Casteen.** He was graduated from the University of Kansas School of Medicine in 1955 and has since been doing postgraduate work at the Indiana University Medical Center, Indianapolis.

The United States Junior Chamber of Commerce (through Mr. Robert V. Cox, president, Tulsa, Oklahoma) is now accepting nominations for 1959 awards to the nation's "ten outstanding young men." Last year five of the ten named, all under 36 years of age, were physicians, recognized for "exceptional achievement in their chosen field."

COUNTY SOCIETIES

The first fall meeting of the Shawnee County Medical Society was held at the organization's office building on September 2. An attorney, Mr. Charles Fisher, Topeka, discussed "Current Trends in Malpractice Cases."

Members of the Wyandotte County Society met at the Town House Hotel, Kansas City, on September 16. Dr. Harold Schnaper, of the Veterans Administration Hospital, Washington, D. C., spoke on "Hypertension."

A meeting of the Golden Belt Medical Society was held at Irwin U. S. Army Hospital, Fort Riley, on October 9 with Col. Milford T. Kubin, M.C., as host. A tour of the hospital started the day's program, after which the staff presented a discussion on gastric lesions. Dinner at the Rimrock Room concluded the event.

Dr. A. A. Fink, Topeka, was speaker at a meeting of the Marshall County Society held at Marysville recently. His subject was blood diseases.

Research Grant to K.U.

Seven contracts totalling $256,986 for research on improved methods of testing possible anticancer agents and for related work were announced in September by the Public Health Service. Included was a grant of $18,128 to the University of Kansas, Lawrence, for maintaining a nucleus colony of inbred mice. This will be a reserve colony so that breeding and experimental mice will always be available.

The contracts will be administered by the Cancer Chemotherapy National Service Center at the National Cancer Institute, Bethesda, Maryland.

Cancer, the second leading cause of death in this country, will strike an estimated one out of every four Americans now alive unless new preventive measures are found, according to the publication *Patterns of Disease* prepared by Parke, Davis and Company for the medical profession.

For a man, chances of developing the disease are one in five, declining after the age 50; and of dying from it, one in eight, declining after the age of 55, says *Patterns.* For a woman, chances of being hit by cancer are one in four, declining after 30; and of dying from it, one in seven, declining after 35.

Economics in Medicine

Effective Credit Policies

FLOYD F. WEHRENBERG, ***Kansas City***

After you have established a standard fee schedule and procedures for explaining those fees to your patients, your next thought will be of your credit policies. It is important that the patient be informed of your charges; it is equally important that you be informed of his paying ability and habits.

Setting up standard policies for the extension of credit will be of further convenience to you since you can then honestly say to your patients that "It is the policy of our office. . . ." This tells Mr. Jones that he is being treated on the same basis as other patients and those requests you make of him will not seem to be discriminatory.

Rating Patients

Obviously, the best way to keep your collections high is to avoid letting any account become delinquent. It is well known that there are many patients who will always take care of their accounts promptly. However, there are those who need encouragement and others you know who will shirk their responsibility if you permit it.

To be certain that each patient is treated properly, it is imperative that you know *in advance* of extending credit what his paying habits are. For this reason we recommend obtaining credit reports on all patients as soon as it is established that they will be permanent patients or before any major service is rendered. From the information in the report you can classify the patient as having "A," "B," or "C" rating. Both his financial card and case history should be marked accordingly. The history is always pulled when the patient comes into the office so this will give your assistant the information in advance of his arriving or at least of his departure. And of course you want all financial information on his account card. Since a rating seldom changes, the cards will have to be marked only once in most cases.

An "A" rating would mean that the patient could be extended unlimited credit (within reason) without concern about his paying. A "B" informs your assistant that arrangements for paying should be worked out with the patient on any major item and that he should be encouraged to pay all charges as he goes along. A "C" would warn you that the patient must be kept on a current basis.

Assume that the "A" patient has just received service and presents the charge slip to your assistant. She thanks him politely and, if he asks about paying, she of course replies that she will be glad to accept payment at that time. (She never indicates she doesn't care or suggests he wait.) However, if he says nothing about paying, neither need she because she is confident he will take care of it at the end of the month.

When the "B" patient presents his charge slip, your secretary says, "Thank you, Mr. Jones. The charge is $10. Would you like to take care of it today?" If he says no, then her reply is, "If you prefer to wait until the end of the month, that will be perfectly all right, Mr. Jones." In this way she imputes to him a promise to pay at the end of the month.

To the "C" patient your secretary will say, "Thank you, Mr. Jones." Then reaching for her receipt book she will add, "The charge is $10 for today." This is a polite way of saying that she is expecting payment at that time. If he does not intend to pay, he will have to explain. She should then reply, "When do you want to take care of it, Mr. Jones?" If he says he'll pay next time or at the end of the month, your secretary has his promise. She will answer, "All right, Mr. Jones. I'll note that on your card." If she can do so in his presence, so much the better. He is thereby bound by his own words and he knows they have been written down.

Your assistant should never miss the opportunity to say, "It is our policy. . . ." New patients should be informed that "It is our policy to send statements at the end of each month." In making financial arrangements, it is well to say "It is our policy to make advance arrangements for the convenience of our patients." This lessens the likelihood of offending.

If you deviate from standard procedure, let the patient know he is an exception. For instance, if he is given a longer time to pay than you ordinarily would give him, say "Under the circumstances, we will do so this time." If he knows you are being lenient because you believe the reason is justified, he will be less likely to ignore his obligation or to tell others that you don't have any policy for enforcing payment.

Your credit policies should be aimed at educating patients to prompt paying habits. If your patients develop such habits, they will stand you and him in good stead even when economic conditions are unfavorable. Therefore, be certain that you set up effective credit procedures in your office and follow them faithfully.

Mr. Wehrenberg is Missouri-Kansas Manager, Professional Management Midwest, 4010 Washington Street, Kansas City, Missouri.

Social Security

The Physician's Role in the Disability Program

Doctors, hospitals, institutions, and agencies having contact with disabled people are frequently asked to fill out medical reports in connection with claims under the disability provisions of the social security law. Under these provisions, disabled workers 50 to 65 years of age, and the disabled dependent sons and daughters of retired or deceased workers, may receive monthly disability payments. Disabled workers under age 50 may have their social security records "frozen." This protects the future benefit rights of the disabled worker and his family.

To qualify under these disability provisions, a person must be unable to engage in any substantial gainful activity by reason of a medically determinable physical or mental impairment which can be expected to result in death or to be of long-continued and indefinite duration. A disabled worker must, in addition, have social security credits for work in at least five out of the ten years before he becomes disabled, including a year and a half out of the three years before his disability began. A disabled child must be both unmarried and dependent, and must have become disabled before his or her 18th birthday.

Applications under the social security disability provisions are taken by the more than 570 social security district offices, located in communities all over the nation. The social security district office gives the disabled applicant information about his rights, helps him to fill out his application, and to get the proofs and documents he may need to support that application. Under the law, the disabled person is responsible for furnishing, at his own expense, the evidence to show that he is "disabled" within the meaning of the social security law.

His social security district office gives him one or more copies of a medical report form on which this evidence can be supplied. He is asked to take or mail this form to his attending physician or to a hospital, institution, public or private agency where he has been treated for his disabling condition. This report form, designed as a guide for the reporting physician, lists the kind of medical facts essential for the determination of "disability." However, the reporting doctor is not required to use it; if he prefers, he may make his report in the form of a narrative summary or he may submit photocopies of the pertinent medical records. The completed reports are to be returned by mail to the social security district office (or to a state agency, if indicated).

By providing a full and objective clinical picture of his patient, the reporting doctor fulfills his responsibility to his patient and, incidentally, expedites the decision. To be of maximum use for the evaluation of a patient's capacity for work, the report should include a history of the impairment, the symptomatology, clinical findings and diagnosis. Obviously, the reporting physician has an important role in the operation of the social security disability provisions. He is not, however, asked to decide the issue of "disability." The determination as to whether a patient is "disabled" must be made within the scope of the social security law; often it is based on evidence from more than one medical source. Also the determination must take into account factors which are not purely medical—factors such as education, training, and work experience.

After the applicant has filed his claim under the disability provisions, and furnished the supporting evidence, his case report is forwarded by his social security district office to an agency of his state—usually the state vocational rehabilitation agency. Under agreements between the individual states and the federal government, these state agencies make the disability determinations for their own residents.

In the state of Kansas, the agreement with the federal government provides for the Kansas State Board of Vocational Rehabilitation to make these disability determinations.

The evaluation of disability is made by a "review team" in the state agency. There are at least two professional people on each team. One of the two is a doctor of medicine (often a practicing physician who serves with the state agency on a part-time basis); the other is trained in evaluating the personal and vocational aspects of disability. The team must decide whether the applicant is sufficiently disabled to prevent him from engaging in any substantial gainful activity within the foreseeable future.

In many cases it is necessary to write back to the reporting physician because the medical report does not contain enough clinical facts. As a rule, the kinds of medical facts that the attending physician needs in making his diagnosis and in treating his patient are the same as those required to evaluate the severity of impairments in disability programs. However, certain medical facts are more highly significant in disability evaluation than in medical management of the case. To evaluate the effect of the impairment on the individual's ability to work requires the kind of medical evidence that confirms the diagnosis and measures remaining functional capacities of mind and body. By furnishing complete and objective evidence, the reporting physician makes it unnecessary for the

reviewing physician to "write back" for additional clinical or laboratory data.

If the medical evidence initially submitted indicates a reasonable likelihood that the applicant is disabled, but more precise clinical or laboratory findings are needed to arrive at a sound decision, or to resolve conflicts in the evidence, a consultative examination (usually at the specialist level) may be ordered to obtain additional information. Selection of consulting physicians and payment of fees are governed by state practices.

Some doctors feel that they should be reimbursed by the government for the cost of preparing the medical reports on their patients, and it is, of course, quite within their prerogative to charge the patient a fee for that service. However, under the law, the Social Security Administration cannot pay that fee; that is the individual's responsibility.

Evaluation of Disability

The central purpose of disability evaluation is to determine remaining mental and physical capacities: (1) what the claimant has left, and (2) what he can do with what he has left.

A realistic evaluation of disability must be based on clinical and laboratory tests of the individual's ability to meet the metabolic demands of activity, to reason, to perceive, and to perform certain basic activities such as sitting, standing, bending and walking. When incapacity results from severe impairment of one or more such functions, it is essential to establish not only the fact that functional impairment exists, but also its extent.

A brief discussion of disability from heart disease may serve to illustrate the kind of evidence needed to measure the patient's remaining functional capacity, after appropriate therapy. Most frequently, impairments of the circulatory system produce loss of bodily function by reduction of cardiac reserve, or interference with peripheral vascular circulation. As a result the circulatory apparatus cannot meet effectively the metabolic demands placed upon it. The diagnosis of the condition usually reflects whether the impairment is caused by valvular disease, myocardial damage or vascular pathology.

Cardiac size by x-ray or physical and electrocardiographic findings furnish objective proof of cardiac pathology. The amount of dyspnea or angina described in terms of the number of steps that can be mounted or distance in feet or blocks that the patient can walk is highly significant to evaluation of the degree of loss of function. The presence or absence of cardiac edema and response to therapy are also indicative of severity of cardiovascular impairments. The status of the pulse in the peripheral vessels may provide gross clinical evidence of impaired circulation of the extremities.

Impairments of the cardiovascular system may manifest themselves with dramatic suddenness, e.g., myocardial infarction, obstruction of vessels in peripheral or central nervous system circulation, lungs, and other visceral organs. The initial clinical manifestations are severe and the prognosis dubious. With survival from the acute stage, and appropriate therapy, substantial improvement can be expected over a period of time. A realistic evaluation of remaining function should be made after the convalescent period. Hence, the clinical and laboratory findings after maximum improvement from treatment are particularly valuable in making a determination of remaining cardiac, brain or other function. (Note that a "waiting period," is prescribed by law, i.e., the first monthly disability insurance benefit cannot be paid until the seventh month after the onset of the disability.) A description of the acute attack helps confirm the diagnosis and should therefore be included in the report.

Loss of function is evaluated on the basis of clinical and laboratory findings after maximum benefit from treatment. Clinical information concerning nature and response to treatment furnishes information on stability of functional capacity, i.e., a history of periodic decompensated heart disease, in spite of treatment, would indicate a comparatively severe condition.

More complicated tests of vascular function may be required in certain cases, e.g., arteriography. The reporting physician should not be concerned because he may not have equipment to perform these tests. A carefully performed exercise tolerance test (if not medically contraindicated) will almost always provide the clinical evidence needed to evaluate the degree of remaining function.

Conclusion

In developing evaluation guides for the use of state agencies and its own technical and professional personnel, the Social Security Administration has had the continuing cooperation of a Medical Advisory Committee appointed by Commissioner Schottland, in February 1955. The committee is composed of recognized specialists associated with medical and allied professions in various fields outside government, such as general practice, research, medical education, industry, and labor.

The American Medical Association has cooperated with the Social Security Administration by informing its members about the medical aspects of the disability program, especially the preparation of medical reports. On June 1, 1957, the *Journal of the American Medical Association* carried a comprehensive report on the administration and organization of the disability provisions. Regulations on the meaning of disability appeared in the September 28, 1957, issue.

BCG

After 35 Years—A Disaster or a Triumph?

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Purpose

Approximately 35 years have passed since the first BCG vaccine was administered. BCG has been administered to 145,000,000 people. Yet it remains a controversial topic in tuberculosis control.

BCG provides a close similarity with another controversial topic in disease control of today, the "Salk vaccine." Here again we are dealing with living, "attenuated" organisms for disease control. We review the history of BCG with the hope that it may help to avoid similar errors in our present practice of medicine.

History

Tuberculosis was once unquestionably the greatest single cause of disease and death in the western world.[17] Five outstanding discoveries recorded in medical history have greatly increased our knowledge of tuberculosis.[9]

1. The demonstration by Auenbrugger in 1761 of a method of percussion by which the comparative densities of subadjacent parts could be determined.
2. The stethoscope described by Laennec in his book published in 1819.
3. The identification of the tubercle bacillus by Koch in 1882.
4. The demonstration of Koch's phenomenon in which the presence of this germ in the body caused the tissues to become sensitized, and the subsequent use of the tuberculin skin test in detecting infected individuals.
5. The discovery of x-ray by Roentgen in 1859.

Increased knowledge of tuberculosis gave impetus toward control of this disease. Since the discovery of tubercle bacilli by Koch, specific immunizations against tuberculosis have been repeatedly attempted.[43] All methods were discarded with the exception of BCG.

The bacillus of Calmette and Guerin (BCG) was isolated in 1902 by Nocard[23, 37] from a tuberculous mastitis in a heifer. One ten-thousandth of a milligram of this virulent culture of bovine tubercle bacilli killed a guinea pig weighing 400 grams in 60 days. Beginning in 1908, Calmette and Guerin at the Pasteur Institute of Lille, France, transplanted this culture on artificial media.

Calmette[23, 32, 37] planted a strain of this bovine tubercle bacillus on ox-bile-potato media. It was noted at that time that beef bile acted like soap, wetting the surface of the bacilli and making it possible to dissociate the organisms into almost single units. After this strain had been transplanted 230 times, the bacilli were avirulent but were still capable of sensitizing to tuberculo-protein the tissues of the animal into which they were introduced. After the first few transplants there was an increase in virulence. The development of this avirulent strain took 13 years. This strain became innocuous for calves and later lost its virulence for monkeys, and, after five years, for all laboratory animals.

The bringing about of this process may be attributed to mass mutation due to the presence of bile. Another possibility is that bile may have inhibited virulent organisms but allowed attenuated ones to grow, much the same as is suspected in the case of the addition of INH to growing cultures of tuberculosis.[37]

At this stage of development Calmette declared that it was a "virus fixe" and gave it the name "BCG" (Bacille Calmette-Guerin).[23] Experiments were begun in protection of animals, Guerin applying himself especially to cattle. By 1922 the results had been so uniformly successful that Weill-Halle ventured to give vaccine to infants.

BCG was first given by Calmette[35] per os in a teaspoonful of milk. The first dose was given within 24 hours after birth with two more doses 48 hours apart. Each dose contained 400,000,000 bacilli. This first vaccination was used a few days after delivery on an infant whose mother died of tuberculosis and who was reared by a grandmother who had an active case of tuberculosis.

Two early events that occurred in regard to BCG[44] led to a reluctance of physicians to accept the vaccine, especially in the United States.

In 1927 Petroff[26] shook the over-enthusiastic confidence in the new BCG vaccine by claiming that it was possible to dissociate a virulent type of colony. Petroff and his associates[44] dissociated BCG into rough and smooth strains and claimed that the smooth strain had the ability at times to produce progressive disease in animals. It is not possible by colony mor-

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This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Ikezaki is now serving his internship at Queen's Hospital, Honolulu, Hawaii.

phology to differentiate unequivocally virulent from attenuated strains of the tubercle bacillus.[37]

Following increasing antagonism to BCG came the disaster in Lubeck, Germany, in 1930 [32, 37] which caused the entire suspension of this vaccine in some countries.

On December 9, 1929 [18, 26, 32, 37] a child was supposedly vaccinated with BCG, and on December 30 a second child received the same dose of these organisms. This was the beginning of a project in which half of the newborn children were to be vaccinated. The child who received his first dose on December 9, 1929, developed tuberculous cervical adenitis which was thought to be on a congenital basis. The child to whom the material was administered on December 30, 1929, remained in good health. The vaccine was thought to be entirely safe, and the project was begun. Between February 28 and April 28, 1930, about one-half (approximately 250) of all babies born in Lubeck received the usual doses of BCG by mouth during the first ten days of their lives.

Myers[32] writes, "A child died on April 17 who had been vaccinated only one month before. On April 20, 1930, a second child died and since the disease was most extensive in the lungs, and the vaccine had been administered orally it was assumed that it could not have been responsible and that the infection had probably occurred through the respiratory tract from exposure to the tuberculous mother. On April 25 and 26 two more children died, whereupon the vaccination project was immediately stopped. These supposedly vaccinated children continued to die until a total of 75 was reached on January 29, 1931. A number of others were definitely ill from tuberculosis."

Myers continues, "The vaccine was kept in a laboratory where there were also cultures of virulent strains of tubercle bacilli. Unfortunately, as soon as it was found that children were falling ill and dying, the physician in charge of the laboratory had all the cultures destroyed in order to prevent any possible use of this material. Thus it was impossible to study the organisms in the containers from which the children were vaccinated. However, organisms obtained at postmortem from the children dead from the vaccination revealed in most of them virulent bacilli thought to be the human type. From this it was deduced that either the original vaccine had been contaminated with virulent organisms or at least some of the children had received inoculations from other containers. . . ."

The trial of Doctors Altstaedt, Deycke, Klotz and Nurse Schutze began on October 12, 1931.[19] They were charged with responsibility for the deaths or bodily injury of infants who had been fed BCG. During Dr. Altstaedt's hearing he stated that he was still convinced of the harmlessness of BCG. He therefore officially recommended the feeding of BCG in Lubeck and saw no reason why he should not do this. He enlisted the assistance of Dr. Deycke, who assumed responsibility for obtaining a culture from Paris and by culture could supply the necessary emulsions. He was assisted by Dr. Klotz and Dr. Jannasch.

The prosecution charged that these workers paid no attention to reports in the literature warning against the feeding of BCG. In addition, the prosecution charged that Dr. Altstaedt failed to heed the Reich regulations of 1911 in regard to the use of vaccines and drugs.

It was assumed that Dr. Altstaedt was aware that for emulsions for the feeding of infants, cultures were also used which, contrary to the recommendations of Calmette, had been cultured on Hohne egg medium, which is supposed to increase the virulence of the cultures.

Dr. Altstaedt tried to show that recent literature supported his assumption that BCG was harmless and no longer supported the position taken by the Health Department of the Reich in 1927. He referred to the work of Lignieres, Lange, Berger, Heimbeck, Tzechnowitzer, Mehlis, and Buschmann. Buschmann had fed 205 infants with BCG with encouraging results.

Dr. Deycke was next heard. He was accused of having wanted to carry out experiments in Lubeck using human tubercle bacilli. This he sharply denied. Dr. Deycke stated, "In 1929 I was firmly convinced that the Calmette procedure was not only non-injurious but also useful. In the time since the Lubeck catastrophe I have come to realize on the basis of my own work and that of other investigators, particularly Petroff, that BCG-vaccine is not harmless."

He stated that he deeply regretted the misfortune which he found hard to bear after having devoted 30 years of work to the problem of tuberculosis control. He pointed out that not less than 208 authors had expressed themselves in favor of the immunizing effect of BCG. He attributed the catastrophe to a spontaneous increase in virulence of the BCG. He did not think a mistake in the cultures used or contamination had occurred in his laboratory. He had a long argument with Professor Ludwig Lange regarding the finding of human bacilli in the vaccine material.

Professor Klotz admitted that in the articles which he published in Medizinische Welt in May and June, 1930, he made serious errors especially in regard to figures published.

Professor Schurmann demonstrated the pathologic anatomy of tuberculosis and its variable form in the 53 children on whom he had performed autopsies. In 97.9 per cent of cases of feeding-tuberculosis which could be demonstrated almost exclusively in

the cadavers, the primary complex of the infection was found in the small intestine, in 75.5 per cent in the neck region as well, in 17 per cent also in the middle of the digestive canal or in the region of the esophagus, in 17 per cent also in the lungs. There were nearly always multiple primary infections. The cause of death in the great majority of cases was specific meningitis. On April 26, 1930, the autopsy findings in a child fed with BCG were demonstrated and showed the damaging effect of the inoculation material. The difference of opinion among physicians in regard to the effect of BCG did not make the best impression on the lay public.

In spite of great efforts on the part of all concerned to throw light into the darkness of the Lubeck castastrophe, little progress was made. No light was shed on the question of possible contamination of cultures in the laboratory. It became apparent that the puzzle never would be solved completely.

Dr. Altstaedt was convinced that the Lubeck vaccination material had been contaminated.

Kolle stated that since the effectiveness of the substance had not been demonstrated, it should not have been used for protective-feeding. That the use of hematin medium for culture purposes in Lubeck had caused a change in virulence was denied. Others rejected the idea of "virus fixe" but saw latent danger in BCG because of the possibility that for unknown reasons the bacillus could again become virulent.

In regard to the question as to whether BCG is harmless or injurious, no agreement could be reached.

Lange and Hahn[19] reported that the virulent bacilli found in the emulsion was identical with a human virulent strain which was obtained from Kiel at that time and which had been used in the laboratory for the preparation of partigen. They assumed, therefore, that contamination of the emulsion had taken place. The Kiel strain had in common with autopsy material cultures a peculiar growth on liquid media and a peculiar green coloration of a chloroform extract of the medium. It was brought out, however, that Professor Schurmann at that time was given the wrong autopsy material in one case. This confused the issue except to emphasize the negligent manner in which the pathologic institute of the hospital was run.

After 75 days of court session the verdict was read. The sentences were two years imprisonment for Professor Deycke and 15 months imprisonment for Dr. Altstaedt. Dr. Klotz and Nurse Schutze were found not guilty. The court decided that contamination of the vaccination material must have occurred in the Deycke laboratory although a spontaneous change in virulence of BCG was thought to be theoretically possible. In support of this assumption were findings that demonstrated identity between the injurious bacilli and the Kiel strain.

Dr. Deycke, who had specialized experience in the study of tubercle bacilli for many years, was found guilty of the death of 68 children and the bodily injury of 131 additional children by carelessly distributing his vaccine material.

Dr. Altstaedt was found guilty in the same way because he had been responsible for preparation of the vaccine material and for its use. Failure to carry out animal experiments was also considered negligent, whether or not Calmette prescribed it. It was held that continuous controlled animal experiments should have been carried out so that contaminated vaccine material would be discovered before it was fed to infants.

The nurse was freed because it could not be proved that she had committed an error in technic. Dr. Klotz was considered not guilty in the Griese case because it was held that missed diagnoses are not punishable since they cannot be avoided in many cases.

However, the mystery remains. Events of this kind cannot be judged according to the articles of the penal code and belong to the realm of destiny.

Costantini[12] writes, "For four months the most difficult scientific problems which have been studied for decades by thousands of scientists all over the world were argued before a tribunal of laymen who were forced to decide in their ignorance on problems which science has not as yet solved, including bacterial mutations, culture media, allergic phenomena, anaphylaxis and immunity. The laymen had to decide whether spontaneous return of the Calmette vaccine to its orginal virulence is theoretically possible, whether such an event could be ruled out in the happenings of Lubeck."

He continues, "However, it is necessary to draw useful teachings from this error. Vaccination against tuberculosis is a problem of greatest importance but vaccination with living bacilli although attenuated in character is a delicate problem and the results should be evaluated carefully in order to avoid errors. The system must be perfected."

In leaving this historical aspect of the development of BCG and the Lubeck disaster, it may be of interest to recall what Calmette himself had reported in 1932. He stated[8] that BCG is administered orally in infants because of the great permeability of children's intestinal mucosa. Subcutaneous vaccination is preferred only for older children and adults. At the Pasteur Institute about 423,000 vaccinations were made in newly-born infants over a period of eight years. "There was a surprising decrease in fatalities during the first five years of life which were due to meningitis or other forms of tuberculosis." Calmette assumes, on the basis of various experimental work of other

writers, that in addition to a specific immunity, there was a "paraspecific" immunization in his inoculations.

Discussion

Controversies in regard to the value of this vaccine are many. The literature is alive with continual opposition among two schools of thought, one group maintaining that the BCG vaccine is a detriment to tuberculosis control and the opposing group praising its high value.

Myers[31] feels that if BCG were highly efficacious, an abundance of convincing proof should be available after more than a quarter of a century of use and vaccination of many millions of people. It should not be controversial now. In nations where it has been most extensively used he has found no evidence of its effectiveness on tuberculosis mortality and morbidity. Greater reductions in these rates have occurred in parts of the world where BCG has not been introduced.

Myers[32] is not convinced that BCG is a factor in the decline in tuberculosis mortality in some cities where BCG has been used among children, his reason being that greater declines have occurred during the same time in cities such as New York where vaccine or BCG has not been used.

Hayes[22] likewise feels that to date the evidence in its favor has not been sufficiently convincing to justify its general use, although BCG has been administered to 10,000,000 persons during the last two or three decades.

Favorable results obtained in the use of BCG have been reported by many. The first child to be vaccinated with BCG had no ill effects. Pottenger[35] notes that despite the evident exposure of the child to environmental infection, no harm came to him.

In the British Isles, according to Malmros,[29] of 22,413 BCG-vaccinated persons during the period 1942-1947, only one developed tuberculosis pleuritis. This subject was tuberculin negative in spite of two BCG vaccinations.

Another favorable control program headed by General C. F. Sams[40] was inaugurated by the supreme command of the allied powers in Japan. As a part of that program approximately 31,000,000 persons had received BCG immunization up to May, 1949. The control program has resulted in a reduction in the mean annual death rate from 280 per 100,000 population in 1945 to 181.1 in 1948. Analysis of the death by age group indicates that the entire reduction has appeared in the age group immunized with BCG. The death rate in non-immunized groups has not been appreciably reduced during this period. Within the immunized groups the mortality has been reduced by 88 per cent.

BCG vaccination of a rapidly increasing population of negative tuberculin reactors, intimately exposed to tuberculous infection, has become an established public health measure in Norway, Sweden, and Denmark since 1926. Birkhaug[6] states that this interest in BCG vaccination has in no way been allowed to interfere with established anti-tuberculosis measures, such as tuberculin surveys and mass x-ray examination for detection of tuberculous disease and controlled isolation and treatment of open cases of tuberculosis, in order to prevent the dissemination of tubercle bacilli. These vigilant measures have resulted in reducing tuberculosis mortality in Scandinavia from more than 250 per 100,000 population in 1900 to fewer than 70 in 1944. A proportional decrease in tuberculosis morbidity, however, has not been observed.

From an occupational standpoint nurses have close contact with tuberculous patients. In a four-year study[23] of 2,085 nurses who were enrolled in five schools of nursing the annual incidence of pulmonary tuberculosis was 6.7 per thousand. It was also shown that incidence of pulmonary tuberculosis among nurses with a negative tuberculin reaction on admission to school was six times that of nurses who reacted to tuberculin on admission.

Nurses were vaccinated with BCG and after one month were assigned to tuberculosis wards. Those not vaccinated were not assigned to tuberculosis wards and served as controls. Of 142 vaccinated nurses, none developed pulmonary tuberculosis during the period of observation. In 199 unvaccinated controls three nurses developed tuberculosis. Scandinavian work[10] suggests that a strong local reaction to BCG is correlated with a high resistance to subsequent superinfection with the human tubercle bacilli.[18]

Medlar[30, 41] points out the difficulties of assessing the value of vaccination with BCG in experimental animals under controlled conditions. This emphasizes the greater difficulties encountered in man because of the existence of uncontrolled and uncontrollable conditions.

In the United States, according to Pope and Chadwick,[9] two mass procedures unquestionably contributed to the control of tuberculosis during the period 1942-1945: the almost complete elimination of tuberculosis in dairy cattle and the widespread pasteurization of milk.

The efficacy of BCG for cattle has been omitted from the discussion up to this point. However, it was stated above that the rapid elimination of tuberculosis in dairy cattle in the United States contributed unquestionably to tuberculosis control. This was brought about without the use of BCG; thus veterinarians of the United States believe that BCG is unnecessary in control of tuberculosis in dairy cows.

Myers[32] cites the work of Dr. E. C. Shroeder of

the United States Bureau of Animal Industry who conducted experiments on BCG. Shroeder reported: "A number of persons seem to entertain the mistaken idea that the treatment of all dairy cows with BCG vaccine would at once eliminate the danger from tubercle bacilli in the raw milk of tuberculous dairy cattle. They should take into account that the vaccine does not cure tuberculosis, that it does not kill tubercle bacilli in the bodies of infected animals, that it does not prevent tuberculous animals from disseminating tubercle bacilli, that it does not prevent the development of tuberculous disease, that it does not prevent the animals from becoming infected with virulent tubercle bacilli, but that it does interfere with the tuberculin test to distinguish between safe and tuberculous animals."

Holms, according to Myers,[32] has the opinion that tubercle bacilli in milk have served as vaccination against tuberculosis in human beings. Because of the control of tuberculous cattle this "immunizing agent" is being taken away from children, and he brings forth the idea that a substitute must be found in BCG.

However, there are problems in application of the vaccination. Cummins[13] and Birkhaug[7] argue that BCG vaccine is impossible to guarantee as conforming to certain rules for standardization; it is a living vaccine and thus not permanently "countable" like the dead vaccines we permit.

The method of introducing vaccine is also considered. Oral vaccination is uncertain in its effect and no longer recommended.[26] Subcutaneous injections produce too high a proportion of abscesses both locally and in the regional lymph glands. Intracutaneous injection is better, and the percutaneous method has been widely applied.

Vorwald, Dworski, Pratt, and Delahant[45] have shown that BCG organisms may remain viable for at least 18 months in tissues of the guinea pig after they are injected intracutaneously. They point out that the chance of a spontaneous enhancement of virulence of BCG is still a possibility.

In this connection Jensen[32] has pointed out that the virulence of BCG changes and if the strain becomes too virulent, abscesses develop with alarming effects among those who receive it. On the other hand, virulence has been found to decline so that it does not even produce allergy.

Stone and Dufault[43] have also questioned the safety of the procedure, but no instance of virulent infection caused by the attenuated vaccine has ever been proved.

Myers[32] adds that although the statement is frequently made that BCG is harmless and that no case has been reported in which it has acquired virulence in man, there is a scarcity of evidence in the literature to support this statement. The number of lesions removed for biopsy or necropsy from vaccinated persons who have fallen ill or died from tuberculosis to determine this point is almost nil. In addition it is humanly impossible to determine this point.

However, in 1955, four fatal cases of supposedly progressive tuberculosis developed in three children and one young adult who had been vaccinated with BCG.[37] In all cases tuberculosis bacilli were isolated which did not produce progressive disease in guinea pig. Peterson[34] states that in these cases there can be no doubt that the BCG vaccine itself produced a progressive infection and this infection went on to a fatal termination in each case. However, these examples do not represent reversion to a virulent state, but a lack of host capacity to respond to the organism and therefore a progressive infection in spite of the fact that the vaccine is a relatively avirulent vaccine. Modern studies in agammaglobulinemia suggest that these patients were examples of this disturbance or an equivalent state in which they are unable to develop any of the resistance mechanism to infection with tubercle bacilli.

Collinson[11] reports ten cases of development of untoward local reactions following vaccination with BCG. In these cases BCG caused an inflammatory reaction, eventual caseation, and abscess formation in both the inoculation site and the lymph nodes draining the area. These cases required surgical intervention. Histological sections microscopically reveal collections of lymphocytes, epithelioid cells, and Langhan's giant cells, with few areas of caseation, typical of tuberculous granuloma.

In line with this discussion is the Lubeck disaster. Since Petroff's alarming discovery that BCG might be capable of producing progressive disease was eventually discounted, the Lubeck disaster, in which 75 infants out of 250 immunized died of generalized tuberculosis within a few months, was attributed to contamination or replacement of BCG culture by virulent tubercle bacilli. Hence Kayne, Pagel, and O'Shaughnessy[26] believe that BCG is completely safe for human beings.

According to Dubos,[17] it was because of a mistake that the babies were vaccinated with enormous numbers of living, virulent tubercle bacilli of the human type. Those babies that survived developed only minor lesions. When last observed 12 years later, the survivors were still free of tuberculosis.

Hayes[22] believes that the infants in the Lubeck catastrophe were fed the virulent Kiel strain of human type bacilli. Rosenthal, Blahd, and Leslie[39] report that the BCG cultures used in vaccinating the Lubeck children were most probably contaminated by a human strain of virulent tubercle bacilli that Professor Deycke in Lubeck had received from Kiel prior to the Lubeck program. This was ascertained from the fact that cultures of the organisms from the children who died, as well as from a few of the vaccine

ampules salvaged, produced a phosphorescent green color on Sauton's media. The Kiel strain produced the same discoloration, a phenomenon so rare that there could be little doubt as to the similarity of the cultures. In testing more than 200 virulent cultures from various sources, only two showed this discoloration.

One of the primary reasons voiced against the use of BCG vaccination is that it deprives the physician and public health official of the most effective means of early tuberculosis diagnosis. Dubos[17] believes that this would be of little consequence if vaccination gave promise of eradicating tuberculosis completely, but the vaccines now available are not sufficiently effective to justify such hope. Even when practiced under ideal conditions, however, vaccination never succeeds in completely preventing tuberculosis in the vaccinated group. Moreover, duration of immunity that it affords is still uncertain.

Tuberculin[20, 24] is a substance used in a simple skin test that was evolved by Robert Koch in his search for a vaccine or specific which would assist in the cure of tuberculosis. It disappointed its discoverer in that particular, but it has developed into an invaluable aid in diagnosis.

According to Pottenger,[35] BCG and heat-killed bacilli produce humoral antibodies and establish fixed cell sensitivity. This means, in case of reinfection, an increased ability to localize the bacilli and heal the focus; in other words, immunity.

However, a thought expressed by Myers[32] is that the tubercle bacillus is helpless in causing severe destruction of tissues or illnesses in the absence of allergy. Sensitivity of the tissues which results in the tuberculin reaction is the most important ally of the tubercle bacillus.

The theory[44] that a positive tuberculin reaction predisposes to reinfections is not compatible with a belief in BCG vaccination. In contrast the theory that the development of a positive tuberculin reaction indicates some degree of resistance to reinfection does explain the belief that BCG vaccination results in increased resistance.

Myers[32] writes that when Calmette began his work on BCG, our knowledge of the dangers of sensitization of tissues to tuberculo-protein was meager. Indeed, at that time most physicians still believed that a definite tuberculin reaction indicated immunity. Much excellent research has since been conducted on the subject of sensitization, which appears to have proved conclusively that sensitiveness of the tissues is a highly dangerous factor in the development of clinical tuberculosis. Moreover, it has been shown that sensitization is independent of immunity, that one may develop in the absence of the other and that the tuberculin test is not a test for immunity but only for sensitivity.

An important note in BCG efficacy is added by Myers: "Despite the fact that allergy to tuberculo-protein has not been proved to indicate the presence of immunity, it is the production of such allergy by BCG and its identification by the tuberculin reaction that is now used as the criterion of immunity."

According to Kayne, Pagel, and O'Shaughnessy,[26] the allergic reaction is not specific. Organisms other than the tubercle bacillus (B. coli, leprosy bacilli, pneumococci, spirochetes) evoke hypersensitive reaction in a tuberculous animal. They cite the experiments of Shwartzman, Bordet, Freud, and Much who found that intradermal injection of certain tuberculin preparations in guinea pigs gave reactions with filtrates of other bacteria when these were injected intravenously. These "heteroallergic" reactions do not occur unless considerable amounts of foreign organisms are injected. They may be explained by the fact that some non-specific agents used have antigens in common with the tubercle bacillus.

The above authors state that experiments by Pagel, Krehl, and Mathes showed that generalized tuberculosis altered the body in such a way that the response to all sorts of irritating agents was modified, the tuberculous animal being usually more susceptible than the healthy animal.

Kayne, Pagel, and O'Shaughnessy further point out that in tuberculous guinea pigs there is a suppression of the serum anaphylactic shock. The tuberculous foci, especially in the spleen and marrow, modify the formation of antibodies, and the animal responds as if it were not sensitized. Similarly, tuberculous animals cannot be infected with anthrax. All this has apparently nothing to do with specific tuberculosis allergy. The explanation is simply that the presence of foci in the organs alters the excretion of foreign material or toxin and the response to any supervening infection. In the same way tuberculosis may not develop or may be retarded after virulent infection in animals suffering from pasteurellosis or other diseases.

In the light of present knowledge Calmette's original premises seem unfounded. Myers[32] points out that Calmette believed that the bovine type of tubercle bacillus had low virulence for man. Therefore, he used this organism in producing BCG. Recent work has shown that the original premise was in error. Calmette also believed that he had produced a "virus fixe" with reference to virulence. This premise was not true. At that time there was a general belief that satisfactory immunity developed in tuberculosis. However, this premise was also in error, for a dependable immunity does not develop even from the presence of virulent tubercle bacilli in the human body.

Calmette's[35] oral administration is based on the fact that during the early days of life bacteria pass through the mucous membrane of the intestinal tract. The dose is given soon after birth to avoid

the possibility of the child having become infected; for, if infected, hypersensitivity might be developed and might cause the child to react violently.

However, Rich[36] points out the pertinent thought that the prophylactic administration of BCG, or vaccine made of killed tubercle bacilli, to newborn infants, as has been widely practiced, means that antigens are being applied to subjects who, in the light of all available evidence, have far less power of reacting to immunizing antigens of all sorts than have individuals at any other period of life. This should be taken into consideration not only in regard to the question of the proper age period for immunization but also in drawing conclusions as to the immunizing power of these vaccines from results obtained in newborn infants. Used at a later period their efficacy would, in all probability, be much greater.

Malmros[29] states that the newborn become tuberculin-positive after vaccination to the same extent as older children and adults, subsequently remaining positive for several years.

Recently Wagner[46] wrote, "The rationale of the use of BCG vaccine rests upon the observation, now clearly established both clinically and experimentally, that resistance to tuberculosis is acquired following a healed primary tuberculosis infection. In vaccination with BCG vaccine the objective is to substitute for the potentially hazardous and uncontrolled naturally acquired first infection with tuberculosis a benign infection which will convert the tuberculin reaction to positive and give some protection against subsequent infection."

Two of the best known "controlled observations," that of Aronson and Palmer[10] and that of Levine,[28, 32] will be presented. One observation gives favorable results for BCG; the other shows that BCG is of questionable value.

Levine reported on a study which was undertaken in December, 1926, by the Bureau of Laboratories of the New York City Department of Health. The children in this project were from tuberculous families and with few exceptions were placed under observation during the first five years of their lives. The study included 1,011 vaccinated cases and 1,073 controls.

It was observed during this study that there was a tendency to administer BCG to children of the more intelligent and cooperative parents who gave their children better care with reference to feeding, etc., and who followed all instructions more faithfully than non-cooperative parents. This routine was in effect until January 1, 1933, during which time 445 children were vaccinated and 545 were observed as controls.

The tuberculosis mortality for this period follows:

	Number	*Tuberculosis Deaths*	*Tuberculosis Mortality*
Vaccinated Cases	445	3	0.68 per cent
Controls	545	18	3.30 per cent

However, on January 1, 1933, the method of selection was changed, so that alternate children were vaccinated. Under this new method of selection, 566 children were vaccinated and 528 served as controls. The mortality rate for this period follows:

	Number	*Tuberculosis Deaths*	*Tuberculosis Mortality*
Vaccinated Cases	566	8	1.4 per cent
Controls	528	8	1.5 per cent

Thus, by this method of selection, no significant difference in mortality between the vaccinated and unvaccinated children was demonstrated. No change in exposure, in racial distribution, in economic conditions, or in number of cases lost was found to account for the difference in figures between the groups selected by different methods. There was no reason to feel that the vaccine was less active, since the local lesions developed as rapidly and reached the same size in similar periods of time in both groups; and the tuberculin tests developed and responded similarly.

On the other hand, Aronson and Palmer[3, 10] made an observation on North American Indians between one and 20 years of age. The vaccinated and control groups were equally exposed to subsequent infection. The results were as follows:

	Vaccinated Indians	*Control Group*
Total Number	1,551	1,457
Tuberculosis Deaths	4	28
Minimal Pulmonary Tuberculosis	8	20
Moderately Advanced Pulmonary Tuberculosis and Extrapulmonary Tuberculosis	9	48
Pleural Effusion	4	18
Hilar Adenitis	19	99
Parenchymatous Pulmonary Lesion	11	74

In this study vaccination gave some degree of protection against all types of lesions, but particularly against the more serious pulmonary and extra-pulmonary lesions. These results have been universally accepted as proof of the efficacy of vaccination for this racial group of known high susceptibility. A later report stated that there had been 55 deaths among the vaccinated, six of which were due to tuberculosis; there were 109 deaths among the controls, 53 due to tuberculosis.

Aronson[3] writes that from his studies there are indications that the use of BCG vaccine is a practical

and safe procedure giving rise to definite protection against tuberculosis.

Besides these two "controlled observations" there are other reports in the literature of today.

DeFriez et al.,[15] in a voluntary program of BCG vaccination of nurses, found no evidence that BCG offered any significant protection to tuberculin-negative student nurses. In contrast, Dannenbaum,[14] in reporting on BCG vaccination of infants within the first ten days of life, concludes that BCG vaccination during the neonatal period provides considerable, if not absolute, protection against tuberculosis if such children are not exposed to the disease until after the Mantoux test has turned positive. In the same tone Bethoux,[5] reporting a study over a period of ten years in 300 infants of tuberculous parents, concludes that BCG vaccination reduces not only the tuberculosis mortality but also the general mortality of the infants.

Dickie[16] in a report of 16 families in which some children were vaccinated with BCG and others were not, and in which all had been exposed to a known source of infection, states that the protective value of BCG vaccination is striking, both when the source of infection is within the family (with the maximum risk for each child) and when it is outside. Similarly, Gernez-Rieux et al.,[21] after vaccination of 15,000 children with BCG, state that their study also confirms the harmlessness and the great efficacy of vaccination with BCG used by scarification.

Abruzzi,[2] reporting on a survey of incidence among American medical students, reports that BCG represents an additive weapon against tuberculosis, particularly as this disease threatens the non-immune person when he encounters heavy tuberculous infection.

Stilwell[42] reported on a description of the technique of vaccination used and its results with respect to immediate changes in sensitivity to tuberculin. His group included student nurses, more than 90 per cent of whom reacted positively to tuberculin six weeks after vaccination. Lafay-Coletsos[27] reported as follows on postvaccinal surveillance in infants vaccinated with BCG-S: At the sixth month, 158 tests, 134 positive results or 84.8 per cent; at the 18th month, 63 tests, 37 positive results or 58.7 per cent.

Bergqvist[4] made a study of attempting to heighten the effect of BCG vaccination with hyaluronidase. Because a larger local reaction is usually obtained when hyaluronidase is added, he feels that it is possible that the new method of vaccination might bring about an improvement in regard to both degree and duration in the state of tuberculin hypersensitivity.

Rosenthal[38] feels that although some differences in the various strains of BCG vaccine exist, these differences are not significant. With the advent of freeze-dried form of BCG vaccine, it has become possible to completely standardize the vaccine before use; the viability, potency, and safety can be ascertained before it is distributed for human consumption.

BCG has been given to millions of people throughout the world and with time we may be able to see its true effect upon the primary disease process. We may in the future see tuberculosis in an altered form.

Orsini[33] reports an interesting case of a child vaccinated at birth with BCG by scarification because the child's mother had pulmonary tuberculosis prior to the delivery. At the time of the birth of the child she was cured perfectly. This child developed tuberculous meningitis, but the mode of onset was peculiar in that there was an onset of convulsive state during apparent complete health with apyrexia for several days. In spite of the fact that treatment was given late, a cure was obtained.

Irvine[25] estimates that about 145 million BCG vaccinations have now been administered in the world. In the world literature there are four reports of persons who died as the result of BCG vaccination. He states that between 1940 and 1949 in England and Wales alone there were 43 deaths from smallpox vaccination. Keeping the above in mind, if one accepts that BCG vaccine is reasonably safe and that the protection it produces, though not so complete as with smallpox vaccination or diphtheria inoculation, is worth while, why is it not advocated that everyone should be vaccinated? The reason is that, unlike antidiphtheria inoculation, BCG cannot be given to susceptible and immune alike. BCG vaccination, he feels, in the uninfected person is like smallpox vaccination in slow motion; if BCG is given to someone who has already been infected, Koch's phenomenon occurs.

There is no evidence[1] that meets strict scientific requirements demonstrating that BCG effects control of tuberculosis despite the suggestive results of a few studies.

Summary and Conclusion

The literature on tuberculosis control with the use of BCG is discussed, and a brief account of the historical Lubeck disaster is given.

Starting with the work of Falk with tubercle bacilli and the isolation of the bovine tubercle bacilli by Nocard, many workers attempted to find an effective immunizing agent. The result was the work by Calmette and Guerin who produced an avirulent bacillus, now commonly referred to as BCG.

BCG was welcomed with great enthusiasm, but two events checked the spread of its use. These were Petroff's work in dissociating the bacilli into a smooth and rough strain, and the Lubeck disaster which resulted in the death of 75 children. The use of BCG

still continued and the controversy around it still persists.

Techniques of producing BCG and methods of evaluating it vary, depending on such seemingly irrelevant factors as country, state, medical school, and personal beliefs. There seems to be a similarity here to the Salk vaccine which is in its infancy now, relatively speaking.

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BOOK REVIEWS

An Introduction to the Study of Experimental Medicine. By Claude Bernard, translated by Henry Copley Greene. Published by Dover Publications, New York City. 225 pages. Price $1.50.

It would be wonderful if medical literature consisted only of scientific articles of primary importance and fundamental significance. Physicians could then keep abreast of the march of medicine without the necessity of subscribing to dozens of journals and wading through endless trash. Such a state of affairs would exist if all medical writers would study and accept the precepts of Claude Bernard, one of the founders of modern physiology and experimental medicine.

It would be pleasant to be able to report that this classic work of one of the masters could be wholeheartedly recommended as a thoroughly satisfactory, instructive, and pleasant book. As a matter of fact the first 150 pages (parts one and two) deal with the philosophy of experimental reasoning and medical research in an excessively wordy and tedious manner. It is ponderous and repetitious, sounding a great deal like Plato's *Republic* except that it is not so long, and is less entertaining in spite of the fact that it is more sensible.

When one gets to part three the situation is quite different. In this final section the master illustrates the principles of scientific reasoning and experimental medicine with examples from his own works. Here he duly emphasizes the part that erroneous hypotheses and chance observations have played in his work. It is here that the author permits the reader to participate in his thought processes.

It seems probable that the really good investigators realize their potentialities by "doing what comes naturally" and not by reading books like this, and probably fortunately so. On the other hand, most contributors to medical literature or most persons engaged in medical research do not "naturally" adhere to scientific principles in their work. If this group could be forced to follow the rigorously scientific precepts expounded at such great length in this book, medical literature would at one stroke be reduced to a reasonable volume, and improved immeasurably in quality.

This book can be recommended to practicing physicians with the understanding that part three contains the heart of the work—*J.D.R.*

The Management of Childhood Asthma. By Frederic Speer, M.D. Published by Charles C Thomas, Springfield, Illinois. 116 pages. Price $4.75.

"A surgeon repairing a hernia does not invite the family in to assist him. A physician dealing with an asthmatic child, however, must do just that, and his success depends very much on his ability to gain the confidence of the parents and enlist their cooperation. To gain their confidence he must understand how they feel about their child, and to enlist their cooperation he must educate them in the fundamentals of allergy and asthma.

"Parents of allergic children regularly have the misconception that the solving of cases depends primarily on the performance of a long series of infallible skin tests. It is well to remind them that the practice of allergy is much like crime detection, and as the detective learns more from witnesses than from the 'crime lab,' the allergist learns more from the family than from skin tests."

These words by Dr. Speer so adequately express the theme of his manual that the reviewer felt obliged to quote them. Most parents of asthmatic children could understand this writing and would benefit by study of this work, and thus both the ill child and the responsible physician would be secondarily aided. In this fashion the text could be a useful therapeutic tool.—*T.C.H.*

Physician's Handbook. Tenth Edition. By Marcus A. Krupp, Norman J. Sweet, Ernest Jawetz, and Charles D. Armstrong. Published by Lange Medical Publications, Los Altos, California. 488 pages. Price $3.00 (paperback).

For nearly 20 years this handbook, now in its tenth edition, has been a favorite among medical students during their clinical clerkships as well as among internes and residents. It is obviously not a book that was meant to be read straight through, but rather was intended as a reference manual. There are chapters on history taking and physical examination, neurologic diagnostic aids, examination of the heart, peripheral blood vessels, x-ray, electrocardiography, surgery, diet, vitamins, fluid and electrolyte balance, and so on until the 30th chapter on drugs and hormones. Textual material is telegraphic, and the book is dominated by tables. Illustrations are limited to line drawings.

While the *Physician's Handbook* is obviously of primary interest to students and house officers, there is probably no physician who would not frequently find helpful material in it if he kept it handy and made a practice of referring to it.—*J.D.R.*

Handbook of Medical Treatment. Sixth Edition. By Milton J. Chatton, Sheldon Margen, and Henry Brainerd. Published by Lange Medical Publications, Los Altos, California. 568 pages. Price $3.50.

Following in the footsteps of its older brother, the *Physician's Handbook*, which emphasizes problems in medical diagnosis, this handbook on medical treatment was designed to serve as a guide to medical students and physicians in the treatment of their patients after a diagnosis has been established or in the symptomatic and supportive treatment of patients who are undergoing diagnostic workups. There are 23 chapters covering general aspects of medical management, general symptomatic treatment, fluid and electrolyte therapy, diseases of the various organ systems, the treatment of infections and intoxications, etc.

In line with the purpose of the book to present a maximum of factual information in a minimum of space, topics are covered in outline fashion and with extremely brief comments which will probably be more effective as reminders of things once understood but now vague, rather than as an adequate exposition of new material. A physician will be well advised to seek more information in a standard book on pharmacology or therapeutics when this manual does not make the subject entirely clear. In a book of this size there is little room for discussing the "pros and cons" of any subject, and the book was certainly not intended to take the place of larger works which explore problems of therapeutics in more detail. It is hoped that those who use it will not be tempted to accept it as final authority.

The drugs in the handbook are listed by their official names with common or proprietary names given in parentheses when these are considered helpful. The outline presentation is adequately supplemented with numerous tables. One can predict that the handbook will continue to be popular among medical students and house officers, but such a wealth of material as it contains should also be of use to practicing physicians. It is the sort of book which might well be kept in the glove compartment of the physician's car.—*J.D.R.*

How to Live with Diabetes. By Henry Dolger, M.D., and Bernard Seeman. Published by W. W. Norton and Co., Inc., New York, for Upjohn Company. 192 pages. Price $3.50.

This book is divided into three parts. First, diabetes in general is discussed. The second portion deals with the management of diabetes. The third portion deals with special problems in diabetes. The book is written in a very interesting manner and is quite informative.

First, a rather detailed history of the discovery of diabetes is given, and the incidence of diabetes and the role of overeating and heredity in the development of diabetes are discussed. The characteristics of childhood and adult diabetes are pointed out, and the manner in which each type usually develops is explained. The authors give an unusual amount of detail regarding the dietary treatment of diabetes before the discovery of insulin.

The difference in opinion as to the degree of rigidity of dietary control is pointed out and Dr. Dolger seems to be at least moderately liberal on this score. He feels that some physicians are too dogmatic with regard to the patient's diet. For example, he would on the one hand not allow juvenile diabetics to have concentrated sugar such as soda pop, candy, and jams but would permit ice cream, cookies, and puddings.

A rather complete history of the discovery of insulin is given, and the different types of insulin available are discussed thoroughly. A careful program is outlined for the diabetic to follow in daily care of his syringe, the insulin, and the tests for sugar and acetone. The background of the development of Orinase is also explained in considerable detail.

The author's opinions as to the criteria for good control are:

(1) The diabetic must feel well;

(2) Normal weight must be maintained;

(3) There should be just enough insulin and dietary restriction to assure proper utilization of food without producing insulin reactions;

(4) Treatment is simplified so far as possible so that the diabetic can live and work with an absolute minimum of interference from the needs of his disease, and

(5) The diabetic should maintain sugar-free urine or the smallest amount of sugar in the urine possible. Considerable advice to parents and to the diabetic child and the doctor is given. Generally, he believes the child should be giving himself his own insulin and caring for his syringe at the age of eight or nine. The emotional problems which are so common with juvenile and adolescent diabetics are considered.

The book is concluded with a short chapter on possible future developments in relation to diabetes with special reference to research. This book would be of value to both the practicing physician and the patient with diabetes. It should be of especial interest to both patients and doctors who have an unusual interest in the historical background of present day problems. —*T.J.L.*

The Psychology of Medical Practice. By Marc H. Hollender, M.D. Published by W. B. Saunders Company, Philadelphia. 276 pages. Price $6.50.

The authors have made a sincere and successful attempt to answer those questions that are frequently asked of the psychiatrist by the internist, obstetrician, gynecologist, or other specialist with regard to using those psychiatric principles that are applicable to the non-psychiatric patient.

Such questions as whether a given patient should be told that he has cancer and a poor prognosis, and if so, how this information should be imparted to him; how should the young married couple be warned of an obstetrical complication without getting them unduly concerned; how should many of the problems in regard to handling the coronary patient or the family of the coronary patient be approached, what should they be told, how much should they know and what is the best manner in which to impart this information. There are separate chapters dealing with nearly every specialty.

It is refreshing to find a book of this type that was not written for psychiatrists and makes no attempt to make a psychiatrist out of the man working in other fields of medicine. It simply offers those suggestions of psychiatric origin that seem applicable to the non-psychiatrist's discipline in dealing with just ordinary, non-psychiatric patients and helping him make his line of therapy more effective. There are many tools that all of us in medicine use in common. For example, aspirin and hot packs are often used by the surgeon on infections, by the internist on inflamed joints, and even by the psychiatrist on muscle spasm of psychogenic origin. We often use these and other measures purely symptomatically. They reduce the patient's pain, they reduce his anxiety, and increase his general comfort.

This book is aimed at helping the doctor keep the patient's and the family's anxiety at a minimum in order that they may be able to better cooperate with the therapeutic procedures that he has to offer. It is aimed at helping the doctor develop the art of medicine, rather than any particular psychiatric skill, and I feel it is well worth reading.—*T.L.F.*

Modern Clinical Psychiatry. By Arthur P. Noyes, M.D., and Lawrence C. Kolb, M.D. Published by W. B. Saunders Company, Philadelphia. 694 pages. Price $8.00.

This is a standard authoritative text of psychiatry. It is written primarily for those who wish to systematically study clinical psychiatry and neurology, but it should be useful to every physician as a desk reference. The chapter headings are clear and follow the diagnostic and statistical manual on mental disorders. It is well-indexed, and treatment recommendations are practical and specific. This revision includes a chapter on psychiatry and the law.—*P.W.T.*

TV Story on Human Senses

The Bell System Science Series will present its second television program devoted to the working of part of the human body in "Gateways to the Mind," the story of the human senses, which will be seen over the NBC network on Thursday evening, October 23. "Hemo the Magnificent," an earlier program in the same series, was devoted to the story of blood and the circulatory system.

Some 14 different senses are discussed in "Gateways to the Mind" in explaining how the senses function as the channels through which all knowledge of the external world is passed to the brain. Both scientific documentary film and animation are used in the program. Much of this material will be new to the general public.

Dr. Wilder Penfield, Montreal brain surgeon, appears on the program to explain his work in exciting sensations by direct stimulation of the brain. Dr. Hadley Cantril, Princeton University psychologist, discusses some of his experiments in sensory illusions on the telecast.

Dr. George Wald, professor of biology at Harvard, was principal advisor on the production of "Gateways to the Mind," and Dr. Frederick Crescitelli, professor of zoology at U.C.L.A., served as consultant. The program was produced for the Bell System by Warner Brothers and stars Dr. Frank C. Baxter, who has appeared in the four earlier programs of the series. The Scientific Advisory Board, composed of ten American scientists, had general supervision over the production.

After its telecast on October 23, "Gateways to the Mind" will be made available on 16 mm. color film by Bell Telephone companies for group showings to interested organizations.

Cancer accounts for one out of every six deaths in this country annually, according to the publication *Patterns of Disease,* prepared by Parke, Davis and Company for the medical profession. The disease has jumped from seventh place to second in U. S. mortality tables in the last 50 years. If the present rates continue, the annual death toll from cancer will double within the next 50 years, "in part due to an increase in the population."

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

Legislators and the Mentally Ill

We are glad to report that at least one member of the Kansas Legislature has an enlightened social conscience.

Several days ago we again voiced a protest against the medieval and barbarous treatment of the mentally ill in Kansas. Under the law, persons believed to be mentally ill are held in county jails until room is found for them in a state hospital.

Rep. Lawrence D. Slocombe of Peabody said in a letter to the editor: "How would you suggest the Kansas Legislature correct this situation? I am sure the Kansas Legislature does not recommend this type of procedure."

The first step in improving the lunacy laws is to recognize that mental illness is primarily a medical problem. A deranged man is a sick man.

The legislators can quite easily discover the basic principles for more moral and humane legislation. All they have to do is ask for recommendations from the leading psychiatrists, the Kansas Medical Society, the Mental Health Association and other interested groups. Law enforcement officers, who have long been bedeviled by their unwilling role as amateur psychiatrists, and judges could also tell the legislators a thing or two.

It is obvious that sheriffs and policemen are not qualified to care for the mentally ill. It may be necessary for law-enforcement officers to restrain the violently deranged. Their duties should cease at that point. The mentally ill should be cared for in hospitals, not jails.

Much unnecessary suffering, shame and tragedy result from this custom of jailing the disturbed. Not long ago, a pretty little high school girl was held in jail for many days. The child was rational most of the time. She was overwhelmed with panic, shame and confusion at being locked up. The matron did her best to comfort the girl. Nevertheless, the patient's mental condition rapidly worsened because of the jail experience.

Kansas lunacy laws reflect the medieval notion that insanity is somehow willful wrong-doing. No enlightened person believes that today. Why does the Legislature continue to keep legislation that nobody wants and that is directly opposed to the public interest?—*Wichita Beacon, September 4, 1958.*

Medical Care

Reno County is losing the physician who has been employed full time to care for those on the welfare rolls. It is unlikely that a successor can be found. There are too many patients for a single doctor to minister to adequately. The rewards for caring for that large a clientele in private practice are much greater.

So Reno County, whether any one prefers it or not, will be forced into some form of socialized medicine for safeguarding the health of its welfare clients. A contract likely will be made with the County Medical Society or a group of its members for them to provide the service for a monthly per capita fee.

Far from being objectionable, such an arrangement should result in improved medical care for those on various forms of relief. They will be ministered to by physicians of their own choice. The better care will naturally have a higher cost, but that is inevitable.

This introduction of socialized medicine, under whatever other name it may be called, however, raises questions involving an issue of long debate. If it is desirable to levy taxes to pay for the medical care of those on the welfare rolls, and for the military and their dependents, is it undesirable to levy taxes to pay for the care of everyone?

If the solution of the problem is better found through voluntary health insurance programs, should not Blue Shield and Blue Cross be expanded to cover all necessary health and hospital services rather than only enumerated ones?—*Hutchinson News, September 4, 1958.*

Quack Doctors Can Kill You

False hope is the easiest commodity to sell. Frightened, ignorant and gullible people always dig deep into their jeans to buy a wonder drug, a cure-all or a magic food.

Time was when the quack depended on a crude and colorful pitch. One does not have to be elderly to remember the medicine show. The "doctor" would set up shop in a vacant lot at the edge of the village. Real live Indians with feathers danced to tom-toms. The villagers and farmers, starved for entertainment, came in a multitude.

The "doctor" delivered an eloquent plea for his snake oil or Indian elixir. He promised cure for everything from dandruff to heart disease. His medicine generally was just alcohol to which flavors had been added.

The modern quack uses more sophisticated methods. He talks learnedly of science and research. He tells his victims that he has secret cures, medicines and diets. He implies that there is a sinister plot to keep him from making people well. What would doctors do for a living if everybody had good health?

The horror of quackery is that it gives its victims false hope. They do not seek genuine treatment until it is too late. Worst of the quacks are those who promise cancer cures. Cancer in its earliest stages can often be cured; if the victim wastes time on phony treatments, medical science cannot save him.

How can one tell a quack from a true physician? There are several reliable ways:

1. No reputable physician has secret treatments known only to himself. Physicians share their knowledge. Scientific information is available to all members of the profession.

2. No reputable physician guarantees cures. He merely guarantees to do all in his power to help the patient.

3. No reputable physician offers testimonials from cured patients. Testimonials are always worthless. Most sick people get well or improve, with or without a physician. Those who die cannot tell their opinion of the treatment.

How can the layman determine whether a treatment or diet or health fad is a dangerous hoax or worthless fraud? Well, the best thing to do is to go to a real doctor—one who graduated from a respected school for physicians, who makes no extravagant claims, who is trusted and honored by his patients and the community—and ask him.

Or, if the layman belongs to that tiny minority that "doesn't trust doctors," let him call the Better Business Bureau, an organization which specializes in getting the facts about all sorts of phonies, racketeers and swindlers in the business world.

Hope can keep you alive. But false hope can kill you before your time. Don't be a victim.—*Wichita Beacon, September 14, 1958.*

Hospital Needs of the Aged

The House of Delegates of the American Hospital Association, meeting in Chicago recently, adopted a statement of policy with respect to meeting the hospital needs of the aged. The statement, which supersedes all previous actions taken by the association, follows:

1. The American Hospital Association is convinced that retired aged persons face a pressing problem in financing their hospital care.

2. It believes that federal legislation will be necessary to solve the problem satisfactorily. It has, however, serious misgivings with respect to the use of compulsory health insurance for financing hospital care even for the retired aged.

3. It believes that all possible solutions must be vigorously explored, including methods by which the dangers inherent in the Social Security approach can be avoided.

4. It believes that every realistic effort should be made to meet the hospital needs of the retired aged principally through mechanisms utilizing existing systems of voluntary prepayment. However, it is conceivable that the use of Social Security to provide the mechanisms to assist in the solution of the problem of financing these needs may be necessary ultimately.

5. It believes that any legislation developed to provide for government participation to meet the hospital needs of the retired aged should be so devised as to strengthen the voluntary prepayment systems, and should conform to the following principles:

a. Legislation designed to provide for the hospital needs of the retired aged should provide essential hospital services and should exclude custodial care provided for nonmedical reasons.

b. Government participation should be restricted to persons over 65 who are not regularly and substantially employed. The voluntary prepayment system provides a satisfactory mechanism for the coverage of other persons, regardless of age.

c. Any program in which the federal government participates to meet the hospital needs of the nonindigent aged should emphasize individual responsibility and make the application of a means test unnecessary for obtaining benefits.

d. Such a program should be based on the service benefit principle and should provide benefits sufficiently comprehensive to remove the major economic barriers to hospital care for the retired aged.

e. Such a program should make benefits available through nonprofit prepayment plans.

f. Hospitals should be paid fully for the cost of care rendered.

g. Such a program should not provide services in facilities operated by the federal government.

h. Such a program should provide reasonable criteria to determine the eligibility of hospitals to participate, but the federal government should be precluded from interfering in the administration and operation of hospitals providing the services.

i. Such a program should maintain the free choice of doctor and hospital by the recipient.

j. Such a program should permit and encourage continuous adaptation to new knowledge in the provision of services.

ANNOUNCEMENTS

Pediatric fellowships available to physicians through Wyeth Laboratories, effective July 1, 1959. Annual stipend of $2,400. Information available from Dr. Philip S. Barba, University of Pennsylvania School of Medicine, Philadelphia.

Three conferences during winter season at British Colonial Hotel, Bahamas: Sixth Bahamas Medical Conference, November 28-December 18; First Bahamas Surgical Conference, December 29-January 17; Serendipity Session, January 18-31. Reservations may be made through Bahamas Conferences, P. O. Box 4037, Fort Lauderdale, Florida.

Twenty-fifth (silver anniversary) meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 3-7, 1959. Interim session, November 29-30, 1958, Rochester, Minnesota. Eleventh annual postgraduate course on diseases of the chest, Park Sheraton Hotel, New York City, November 10-15, 1958. Information available from the College, 112 East Chestnut Street, Chicago 11, Illinois.

American Goiter Association offers Van Meter prize award of $300 for best essays submitted concerning problems related to the thyroid gland. Deadline, January 15, 1959. Information available from the association, 149½ Washington Avenue, Albany 10, New York.

Calendar of courses available at University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado: October 30-November 1, Western Institute on Epilepsy; January 19-24, General Practice Review; March 16-21, Medical Technology; May 14-16, Neurology; May 21-22, Colorado Intern-Resident Clinic; June 15-19, Internal Medicine; June 22-27, Clinical Hematology; July 6-8, Obstetrics and Gynecology; July 6-9, Ophthalmology; July 16-18, Dermatology for General Practitioners; August 10-15, Western Cardiac Conference; August 24-26, Prevention and Management of Athletic Injuries; September 10-15, Pediatrics.

International College of Surgeons, 24th Annual Conference of North American Federation, Palmer House, Chicago, September 13-17, 1959. Write Dr. Ross T. McIntire, 1516 Lake Shore Drive, Chicago 10, Illinois.

Department of Medicine, Menorah Medical Center, 4949 Rockhill Road, Kansas City 10, Missouri, announces 1958 A. Morris Ginsberg Memorial Seminar. Dr. Sol Sherry, professor of medicine, Washington University, St. Louis, guest lecturer. Write Dr. Morton C. Creditor, director, Medical Education and Research, Menorah Hospital.

Kansas Board of Basic Science Examiners will give examinations in anatomy, bacteriology, chemistry, pathology, and physiology, November 24-25, Wahl Hall, University of Kansas Medical Center, Kansas City. Applications accepted until 30 days prior to examination. Application blanks available from L. C. Heckert, secretary of board, Pittsburg, Kansas.

Future Needs in Medicine

Expenditures for medical research in this country can and should be tripled to reach a billion dollars a year by 1970, a group of special consultants to the Secretary of Health, Education, and Welfare said in a report released today.

The consultants warned, however, that a medical research effort of this magnitude will require a major increase in the number of physicians and other scientists engaged in medical research—from 20,000 now to 45,000 by 1970.

They also said that funds for construction of medical schools will be needed "on a much larger scale than has heretofore been contemplated" and that even if large funds are provided "it seems certain that the number of physicians per 100,000 population will decline" during the next 12 years.

The report was presented to Secretary of Health, Education, and Welfare Marion B. Folsom, who appointed the group about a year ago to advise him on long-term needs in medical research and medical education.

Ten prominent medical educators and industry research executives participated in the study. Chairman of the group was Dr. Stanhope Bayne-Jones, formerly dean of the Yale University School of Medicine, president of the Joint Administrative Board of the New York Hospital-Cornell Medical Center, and technical director of research, Office of the Surgeon General, Department of the Army.

"The expansion of medical research and education required in the national interest will be costly and should not be restricted by lack of funds," the report states.

"The consultants believe it conservative to project total national medical research expenditures of $900 million to $1 billion per year by 1970, as compared with $330 million in 1957."

But the medical schools of today, the consultants emphasized, cannot turn out enough doctors both to provide sufficient staff for a research program of this scale and to meet growing medical care needs of the expanding population.

The consultants expressed the belief that "it would not be in the public interest for the number of physicians in the nation to fall below the ratio of 132 for each 100,000 persons in the population."

"This ratio," the report points out, "has remained constant (plus or minus two) over the past 30 years."

Maintaining this ratio, the report states, would involve the construction of from 14 to 20 new medical schools at a cost of between $500 million and $1 billion.

Pointing out that there is usually a lapse of about 10 years from the time a school is planned until the first class is graduated, the report explained, "even if funds in the order of $500 million to $1 billion were made available immediately for construction of new medical schools, it seems certain that the number of physicians per 100,000 population will decline between now and 1970. Unless there is a marked change in social philosophy leading to private gifts or state appropriations on an unprecedented scale, large federal appropriations will be required."

The consultants suggested that the federal government continue to provide about half of all funds for medical research. On this basis, federal expenditures would increase from $186 million last year to approximately $500 million by 1970.

Industry's share of last year's medical research costs was estimated at $90 million and contributions of private philanthropy at $35 million, but the committee said that both estimates were probably low. Income from endowments was about $19 million.

The committee's projections call for annual expenditures of more than $300 million by industry and for contributions of more than $100 million by private philanthropy by 1970.

The report notes that the portion of the nation's gross national product spent for research and development of all kinds rose from .2 per cent in 1930 to 2.3 per cent last year and can be expected to rise gradually to 4 per cent by 1970.

Projected increases in expenditures for medical research, the report states, are "based on the assumption that the proportion of all research and development expenditures represented by medical research will remain constant at the average for the past decade—3.8 per cent."

The committee said that according to present estimates there will be only 3,200 additional physicians and 16,000 additional Ph.D.'s available for medical research during the next 12 years—almost 6,000 short of the 25,000 additional trained workers needed to staff a billion-dollar-a-year research program.

However, the consultants said that "it should be possible to produce 20,000 scientists with Ph.D. degrees who will enter medical research" by 1970 and that it is "quite possible that . . . the proportion of physicians who elect essentially full-time careers in research will rise."

The report recommended increases—in many instances "substantial" ones—in virtually all programs of the Department of Health, Education, and Welfare involving medical research.

The report points out that the Public Health Service's National Institutes of Health at Bethesda, Maryland, operates "the largest group of medical research laboratories in the world."

These direct operations, together with the large grant programs which support medical research and training in institutions throughout the country, "have over recent years exerted the most important single influence upon the dimensions of American medical research," the report states.

"The consultants are of the opinion," the report states, "that the system evolved for the administration of research and training grants is sound, and that the system has been operated well."

P.H.S. Grant to Squibb

A grant of $190,063 to E. R. Squibb and Sons, New York, to produce unusual androgens and other hormonal substances, was announced last month by the U. S. Public Health Service as part of a program involving $859,442 allocated to two pharmaceutical firms and five research organizations. A second Squibb contract, amounting to $81,015, is for evaluating hormonal substances and anticancer agents in animals and for seeking new methods of studying interrelationships between tumors and the endocrine system.

One hundred thirty-five medical, dental, engineering, science, nurse and veterinary students from 120 approved four-year professional schools spent the summer months in U. S. Public Health Service facilities under the Service's year-round training program.

The program is known as COSTEP, for Commissioned Officer Student Training and Extern Program. Assignments under COSTEP are open each year to students who complete either their second or third year of professional education on or before July 1 and who are interested in becoming Reserve Officers in the Service's Commissioned Corps.

Two students from the University of Kansas School of Medicine, Joseph C. McDaniel and Robert O. Settle, participated during the summer of 1958.

Technique in Heart Valve Surgery

A technique for evaluating the effects of heart-valve surgery while an operation is in progress has been developed by a team of surgeons and cardiologists at the Public Health Service's National Heart Institute in Bethesda, Maryland.

With this technique, direct measurements are made of the heart's output of blood and of differences in pressure on each side of stenosed valve openings just before and after these openings are surgically enlarged.

The new technique allows a more precise and immediate evaluation of the results of heart valve surgery than has previously been possible with conventional techniques, by which pressures and output of blood are measured before and after an operation but not during it.

By making these measurements while an operation is being performed, the new technique tends to eliminate the influence of all variables except the critical one—the size of the valve opening. It gives the surgeon a better idea of how much of the valve's function has been restored and lets him know whether to enlarge the valve opening still further during the same operation.

The new method is applicable in operations for stenosis of any of the four valves of the heart. Its value was demonstrated in studies of 24 patients undergoing valve surgery at the Heart Institute (18 with mitral, four with aortic, and one each with pulmonic and tricuspid valve stenosis).

The new technique was developed by Doctors Herbert Tanenbaum, Eugene Braunwald, and Andrew Morrow of the Heart Institute Clinic of Surgery.

After exposing the heart to view, the Heart Institute team measures pressures simultaneously in the chambers immediately upstream and downstream from the diseased valve by puncturing these chambers with fine (20-gauge) hypodermic needles. The needles are attached directly to two hand-held pressure transducers, which change the pressure pulses coming through the needles into electrical pulsations.

The pulsations from both transducers are projected as two parallel tracings moving across the screen of a cathode ray oscilloscope. The difference between these synchronized pressure tracings reflects gradient across the diseased valve.

The effect of the surgery in relieving the abnormal pressure gradient is apparent when these measurements are repeated immediately after the surgeon opens the heart and inserts his finger (or an instrument) into the valve opening to enlarge it.

To determine the heart's output of blood just before and after this valve manipulation, the Heart Institute team injects measured amounts of a special blue dye directly into either the pulmonary artery or the left atrium, which carry the blood from the lungs to the left ventricle to be pumped to all body tissues.

As it circulates through the heart and out into the arteries, the dye-containing blood is drawn at a constant rate from an arm artery through a densitometer (a photoelectric device that detects the dye concentration). The concentration of the dye and its transit time through the circulation are also projected visually as a "dye dilution curve." The characteristics of this curving line indicate the output of blood from the heart.

In the past, accurate evaluation of the results of commissurotomy has been dependent on measuring pressure gradients through a flexible plastic catheter tube threaded into the heart from outside the body during the periods before and after the operation. During these comparatively lengthy and eventful intervening periods, factors other than surgery on the valve—such as variations in the patient's medical therapy, stress and rest experiences—can alter the pressure and flow patterns within the heart and thus mar the precision of the evaluation data.

Testing for Fire Hazards

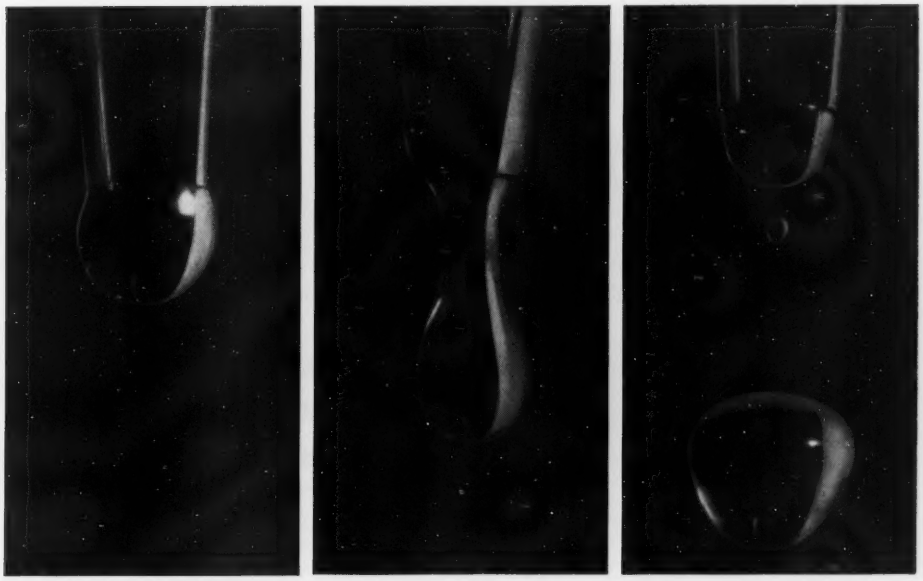
Testing hospital operating rooms for fire hazards is a vitally important safety measure which needs fire department attention, according to *Firemen Magazine,* internationally circulated publication of the National Fire Protection Association.

Writing in the September issue of the magazine, NFPA Gases Engineer Clark F. Jones details methods for fire officers to use in checking hospital operating rooms. The article particularly deals with ways to measure electrostatic charges which can ignite flammable anesthetic agents.

Fire department officers as well as hospital authorities in particular have to be well informed on such fire prevention measures, Jones says, for there are fundamental, important procedures that must be followed for fire safety in operating rooms. His article in particular tells how to use an ohmmeter to test electrical resistance as recommended in nationally accepted standards.

Pedestrian fatalities reached 7,500 and injuries reached 22,000 last year. Over 34 per cent of pedestrian deaths resulted from crossing in the middle of the block, a total of 2,600 persons.

In 1955, 2,158,000 persons were injured in motor vehicle accidents.



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To diabetics and their physicians, CLINITEST means rapid *and* reliable urine-sugar testing—standardized for accurate results every time. And now, the new streamlined model (No. 2105) gives your diabetics this standard test in the best looking, most efficient form.

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Model No. 2105 CLINITEST Urine-Sugar Analysis Set contains everything needed for accurate standardized testing: bottle of 36 CLINITEST Reagent Tablets, test tube, unbreakable dropper. color scale—instruction sheet, analysis record, diabetic's identification card

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Now—the most widely prescribed tranquilizer[1] in sustained release capsules

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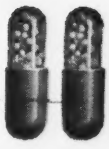

q. 12 h.

Two capsules on arising **last all day**
Two capsules at bedtime **last all night**
relieve nervous tension on a *sustained* basis, without between-dose interruption

"The administration of meprobamate in sustained action form [Meprospan] produced a more uniform and sustained action . . . these capsules offer effectiveness at reduced dosage."[2]

Dosage: 2 Meprospan capsules q. 12 h.
Supplied: 200 mg. capsules, bottles of 30.

1. Meprobamate is more widely prescribed than any other tranquilizer. Source: Independent research organization; name on request.
2. Baird, H. W., III: A comparison of Meprospan (sustained action meprobamate capsule) with other tranquilizing and relaxing agents in children. Submitted for publication, 1958.

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THE FOLLOWING FINDINGS ARE HIGHLY INDICATIVE OF GOUT: (1) *Tophaceous deposits* resulting in irregular, asymmetrical deformity of joints; (2) *Elevated serum uric acid levels* (above 6 mg.%); (3) *Pain relief with colchicine.* When findings suggest gout, therapy with 'Benemid' should be started immediately.

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CAPSULES
(black and white)
250 mg., 125 mg.

ORAL SUSPENSION
(orange-flavored)
125 mg. per tsp. (5 cc.), 2 oz. bottle

NEW! PEDIATRIC DROPS
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glucosamine potentiated tetracycline with nystatin

antibacterial plus added protection against monilial superinfection

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For relief of symptoms and malaise of the common cold and prevention of secondary complications

CAPSULES (black and orange) Ea. capsule contains: Cosa-Tetracyn 125 mg. • phenacetin 120 mg. • caffeine 30 mg. • salicylamide 150 mg. • buclizine HCl 15 mg.

REFERENCES: 1. Carlozzi, M.: Ant. Med. & Clin. Therapy 5:146 (Feb.) 1958. 2. Welch, H.; Wright, W. W., and Staffa, A. W.: Ant. Med. & Clin. Therapy 5:52 (Jan.) 1958. 3. Marlow, A. A., and Bartlett, G. R.: Glucosamine and Leukemia. Proc. Soc. Exp. Biol. & Med. 84:41, 1953. 4. Shalowitz, M.: Clin. Rev. 1:25 (April) 1958. 5. Nathan, L. A.: Arch. Pediat. 75:251 (June) 1958. 6. Cornbleet, T.; Chesrow, E., and Barsky, S.: Ant. Med. & Clin. Therapy 5:328 (May) 1958. 7. Stone, M. L.; Sedlis, A., Bamford, J., and Bradley, W.: Ant. Med. & Clin. Therapy 5:322 (May) 1958. 8. Harris, H.: Clin. Rev. 1:15 (July) 1958.

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Available forms:

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or the treatment of moderately acute infec-ons in infants and children, the recom-ended dosage is 1 teaspoonful per 15 to lbs. of body weight per day, administered 2 to 4 equal doses. Severe or prolonged fections require higher doses. Dosage for ults is 2 to 4 teaspoonfuls 3 or 4 times daily, pending on the type and severity of the in-ction.

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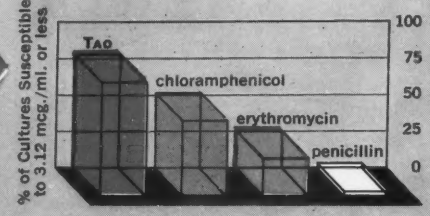
CLINICAL RESULTS	adults	children	all Staph Infections
Cured	172 (80%)	148 (89%)	71 (88%)
Improved	28 (13%)	8 (5%)	7 (9%)
Failure	17 (7%)	11 (6%)	3 (3%)

Types of infecting organisms: The majority of identified etiologic microorganisms were Staph. aureus and Staph. albus. Tao has its greatest usefulness against organisms such as: staphylococci (including strains resistant to other antibiotics), streptococci (beta-hemolytic strains, alpha-hemolytic strains and enterococci), pneumococci, gonococci, Hemophilus influenzae.

even against resistant staph[1,2] . . .

Per cent of "antibiotic-resistant" epidemic staphylococci cultures susceptible to Tao, erythromycin, penicillin and chloramphenicol.[1]



well tolerated . . .

REACTIONS:

(a) adults	(b) children
Total—9.2% (20 out of 217)	Total—0.6% (1 out of 167)
Skin rash—1.4% (3 out of 217)	Skin rash—none
Gastrointestinal—7.8% (17 out of 217)	Gastrointestinal—0.6% (1 out of 167)

There was complete freedom from adverse reactions in 94.5% of all patients. Side effects in the other 5.5% were usually mild and seldom required discontinuance of therapy.

plus . . . stability in gastric acid • rapid, high and sustained blood levels • high urinary concentrations • outstanding palatability in a liquid preparation.

Dosage and Administration: Dosage varies according to the severity of the infection. For adults, the average dose is 250 mg. q.i.d.; to 500 mg. q.i.d. in more severe infections. For children 8 months to 8 years of age, a daily dose of approximately 30 mg./Kg. body weight in divided doses has been found effective. Since Tao is therapeutically stable in gastric acid, it may be administered at any time, without regard to meals.

Supplied: Tao Capsules—250 mg. and 125 mg.; bottles of 60. Tao for Oral Suspension—1.5 Gm.; 125 mg. per teaspoonful (5 cc.) when reconstituted; unusually palatable cherry flavor; 2 oz. bottle.

References: 1. English, A. R., and Fink, F. C.: Antibiotics & Chemother. (Aug.) 1958. 2. English, A. R., and McBride, T. J.: Antibiotics & Chemother. (Aug.) 1958. 3. Wennersten, J. R.: Antibiotic Med. & Clin. Therapy (Aug.) 1958. 4. Celmer, W. D., et al.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 476.

Rx

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SCIENCE FOR THE WORLD'S WELL-BEING

®TRADEMARK

all cold symptoms

New timed-release tablet provides:

...the superior decongestant and antihistaminic action of Triaminic

...non-narcotic cough control as effective as with codeine, but without codeine's drawbacks

...an expectorant to augment demulcent fluids

...the specific antipyretic and analgesic effect of well-tolerated APAP

...the prompt and prolonged activity of timed-release medication

Each* TUSSAGESIC *Tablet contains:

TRIAMINIC®	50 mg.
(phenylpropanolamine HCl	25 mg.;
pheniramine maleate	12.5 mg.;
pyrilamine maleate	12.5 mg.)
Dormethan (brand of dextro-methorphan HBr)	30 mg.
Terpin hydrate	180 mg.
APAP (N-acetyl-para-aminophenol)	325 mg.

Tussagesic Tablets provide relief from *all* cold symptoms in minutes, lasting for hours.

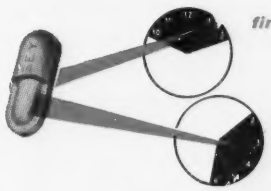
Dosage: One tablet in the morning, mid-afternoon, and in the evening, if needed. The tablet should be swallowed whole to preserve the timed-release action.

To reduce upper respiratory congestion and irritating secretions.

For non-narcotic control of the cough reflex.

To augment demulcent respiratory secretions.

For specific, highly effe ive antipyresis and analgesia.



first—3 to 4 hours of relief from the outer layer

then—3 to 4 more hours of relief from the inner core

Also available—for those who prefer palatable liquid medication— Tussagesic suspension

NEW

Tussagesic

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska • Peterborough, Canada

Low Dosage KYNEX* for G.U. Infections

Sulfamethoxypyridazine Lederle

Unusual Antibacterial and Anti-infective Properties—More soluble in acid urine[1] . . . higher and better sustained plasma levels than any other known and useful antibacterial sulfonamide.[2]

Unprecedented Low Dosage—Less sulfa for the kidney to cope with . . . yet fully effective. A single daily dose of 0.5 to 1.0 Gm. maintains higher plasma levels than 4 to 6 Gm. daily of other sulfonamides—a notable asset in prolonged therapy.[2]

Dosage: The recommended adult dose is 1 Gm. (2 tablets) the first day, followed by 0.5 Gm. (1 tablet) every day thereafter, or 1 Gm. every other day for mild to moderate infections. In severe infections where prompt, high blood levels are indicated, the initial dose should be 2 Gm. followed by 0.5 Gm. every 24 hours.

KYNEX—WHEREVER SULFA THERAPY IS INDICATED

Tablets: Each tablet contains 0.5 Gm. (7½ grains) of sulfamethoxypyridazine. Bottles of 24 and 100 tablets.

Syrup: Each teaspoonful (5 cc.) of caramel-flavored syrup contains 250 mg. of sulfamethoxypyridazine. Bottle of 4 fl. oz.

references:
1 Grieble, H.G., and Jackson, G.G.: Prolonged Treatment of Urinary-Tract Infections with Sulfamethoxypyridazine. *New England J. Med.* 258:1-7, 1958
2. Editorial: *New England J. Med.* 258:48-49, 1958.

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*Reg. U. S. Pat. Off.

When the bronchial tree has too much "bark"

make cough MORE PRODUCTIVE, LESS DESTRUCTIVE

"Significantly superior"[2] cough therapy for "markedly" reducing the severity and frequency of coughing,[1] for increasing respiratory tract fluid,[1] for making sputum easier to raise,[3] and for relieving respiratory discomfort.[4]

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Ethical Pharmaceuticals of Merit since 1878

References:

1. Blanchard, K., and Ford, R. A.: Clin. Med. 3:961, 1956. 2. Cass, L. J., and Frederik, W. S.: 2:844, 1951. 3. Hayes, R. W., and Jacobs, L. S.: Dis. Chest 30:441, 1956. 4. Schwartz, E., Levin, L., Leibowitz, H., and McGinn, J. P.: Am. Pract. & Digest Treat. 7:585, 1956.

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Robins

Antitussive-Demulcent-Expectorant:
Glyceryl guaiacolate 100 mg. and desoxyephedrine hydrochloride 1 mg. per 5 cc.

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Robitussin with Antihistamine and Codeine: Same formula as Robitussin, plus prophenpyridamine maleate 7.5 mg. and codeine phosphate 10 mg. per 5 cc. (Exempt narcotic)

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Recent clinical research demonstrated the excellent value of Supp-hose for leg fatigue, and mild disorders where heavy surgical stockings are not prescribed. The advantage of Supp-hose is that it looks just like any sheer nylon stocking, thus it overcomes one of the main objections of the patient concerned about her appearance.

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the chill

the cough

the aching muscles

the fever

Viral upper respiratory infection.... For this patient, your management will be twofold—prompt symptomatic relief plus the prevention and treatment of bacterial complications. PEN·VEE·*Cidin* backs your attack by broad, multiple action. It relieves aches and pains, and reduces fever. It counters depression and fatigue. It alleviates cough. It calms the emotional unrest. And it dependably combats bacterial invasion because it is the only preparation of its kind to contain penicillin V.

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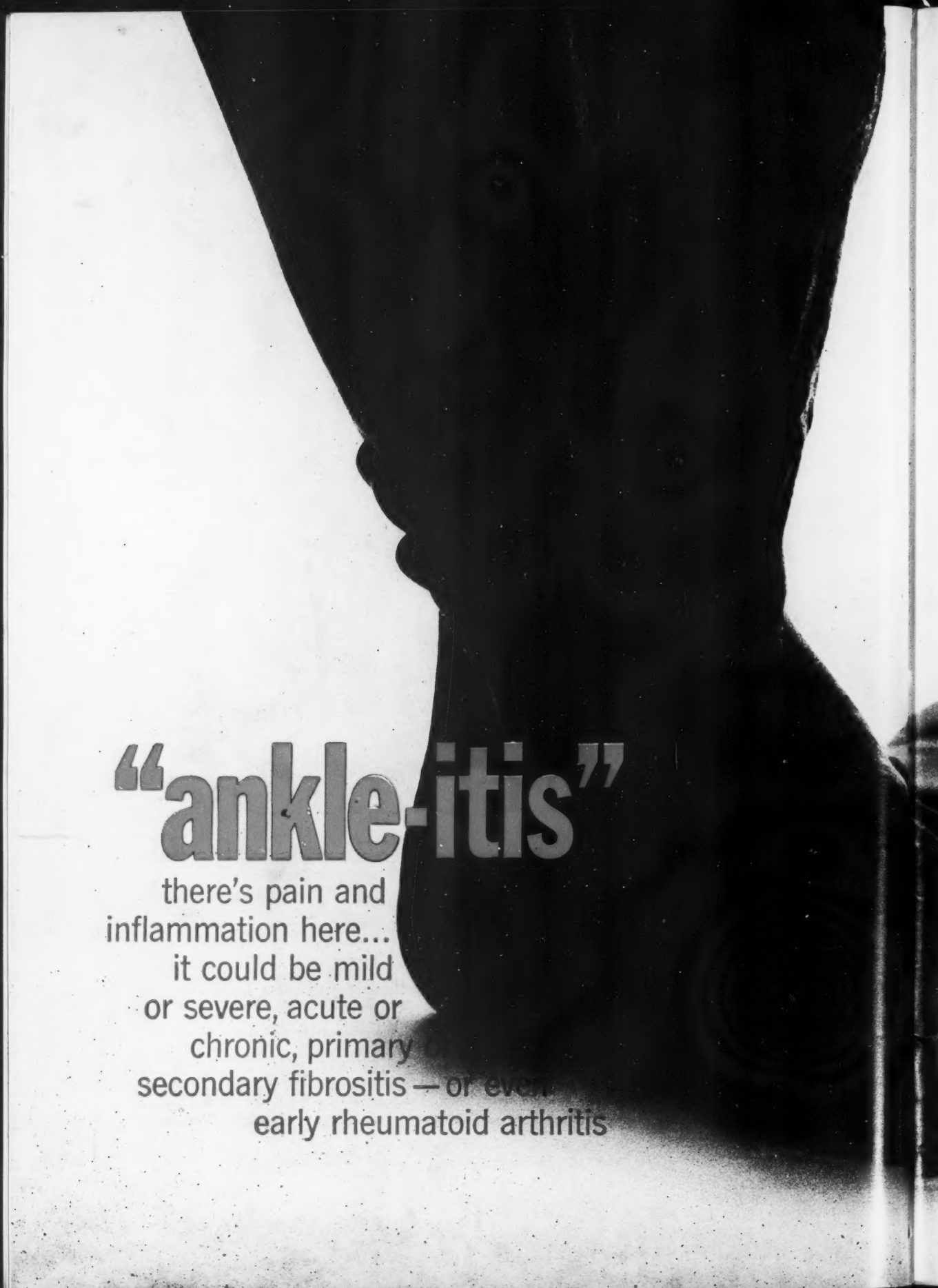
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it could be mild
or severe, acute or
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early rheumatoid arthritis

more potent and comprehensive treatment than salicylate alone

assured anti-inflammatory effect of low-dosage corticosteroid[1] . . . additive antirheumatic action of corticosteroid plus salicylate[2-5] brings rapid pain relief; aids restoration of function . . . wide range of application including the entire fibrositis syndrome as well as early or mild rheumatoid arthritis

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acute conditions: Two or three tablets four times daily. After desired response is obtained, gradually reduce daily dosage and then discontinue.

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precautions: Because SIGMAGEN contains prednisone, the same precautions and contraindications observed with this steroid apply also to the use of SIGMAGEN.

in any case it calls for

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corticoid-salicylate compound tablets

Composition

METICORTEN® (prednisone)	0.75 mg.
Acetylsalicylic acid	325 mg.
Aluminum hydroxide	75 mg.
Ascorbic acid	20 mg.

Packaging: SIGMAGEN Tablets, bottles of 100 and 1000.

References: 1. Spies, T. D., et al.: J.A.M.A. 159:645, 1955. 2. Spies, T. D., et al.: Postgrad. Med. 17:1, 1955. 3. Gelli, G., and Della Santa, L.: Minerva Pediat. 7:1456, 1955. 4. Guerra, F.: Fed. Proc. 12:326, 1953. 5. Busse, E. A.: Clin. Med. 2:1105, 1955. 6. Sticker, R. B.: Panel Discussion, Ohio State M. J. 52:1037, 1956.

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now—an antibiotic troche that

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The *cough control* provided by homarylamine (a non-narcotic antitussive) approximates that of codeine.

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PENTAZETS now extend the therapeutic usefulness of convenient troche medication. Each pleasant-tasting PENTAZETS troche acts promptly against the most bothersome aspects of mouth and throat irritations.

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—the remarkably efficient skeletal muscle relaxant, unique in chemical formulation, and outstanding for sustained action and relative freedom from adverse side effects.

PUBLISHED REFERENCES: 1. Carpenter, E. B.: Southern Medical Journal 51:627, 1958. 2. Forsyth, H. F.: J.A.M.A. 167:163, 1958. 3. Little, J. M., and Truitt, E. B., Jr.: J. Pharm. & Exper. Therap. 119:161, 1957. 4. Morgan, A. M., Truitt, E. B., Jr., and Little, J. M.: J. Am. Pharm. Assn., Sci. Ed. 46:274, 1957. 5. O'Doherty, D. S., and Shields, C. D.: J.A.M.A. 167:160, 1958. 6. Park, H. W.: J.A.M.A. 167:168, 1958. 7. Truitt, E. B., Jr., and Patterson, R. B., Proc. Soc. Exper. Bio. & Med. 93:422, 1957. 8. Truitt, E. B., Jr., Patterson, R. B., Morgan, A. M., and Little, J. M.: J. Pharm. & Exper. Therap. 119:189, 1957.

Supply: Tablets (white, scored), 0.5 Gm., bottles of 50 and 500.

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Ethical Pharmaceuticals of Merit since 1878

Summary of four new published clinical studies:

Robaxin Beneficial in 95.6% of Cases of Acute Skeletal Muscle Spasm[1, 2, 5, 6]

CONDITION	NO. PATIENTS	RESPONSE			
		"marked"	moderate	slight	none
STUDY 1[1]					
Skeletal muscle spasm secondary to acute trauma	33	26	6	1	—
		"pronounced"			
STUDY 2[2]					
Herniated disc	39	25	13	—	1
Ligamentous strains	8	4	4	—	—
Torticollis	3	3	—	—	—
Whiplash injury	3	2	1	—	—
Contusions, fractures, and muscle soreness due to accidents	5	3	2	—	—
		"excellent"			
STUDY 3[5]					
Herniated disc	8	6	2	—	—
Acute fibromyositis	8	8	—	—	—
Torticollis	1	—	—	1	—
		"significant"			
STUDY 4[6]					
Pyramidal tract and acute myalgic disorders	30	27	—	2	1
TOTALS	138	104 (75.3%)	28 (20.3%)	4	2

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"In the author's clinical experience, methocarbamol has afforded greater relief of muscle spasm and pain for a longer period of time without undesirable side effects or toxic reactions than any other commonly used relaxants . . ."[2]

THE JOURNAL
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"An excellent result, following methocarbamol administration, was obtained in all patients with acute skeletal muscle spasm."[5]

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"In no instance was there any significant reduction in voluntary strength or intensity of simple reflexes."[6]

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"This study has demonstrated that methocarbamol (Robaxin) is a superior skeletal muscle relaxant in acute orthopedic conditions."[1]

proven effective
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■ does not impair mental efficiency or physical performance ■ relieves both mental and muscular tension ■ does not affect autonomic function

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9, April 1956. 179. Friedman, A. P.: The treatment of chronic headache with mepro- Use of tranquilizers in the treatment of headache. Am. Pract. & Digest Treat. 8:94, .M.A. 163:1111, March 30, 1957. 182. Friedman, H. T. and Marmelzat, W. L.: Adverse robamate [Miltown]- a clinical study. Am. J. Psychiat. In press, 1957. 184. Gibbs, Relaxant effects of meprobamate in disabilities resulting from musculoskeletal and 453, July 1956. 186. Gillette, H. E.: The effect of meprobamate on cerebral palsy- ers and purpura hemorrhagica (Correspondence). J.A.M.A. 161:96, May 5, 1956. 188. An evaluation of meprobamate in the treatment of alcoholism. Ann. New York Acad. Sc. "Habit-forming" individuals (Queries and Minor Notes). J.A.M.A. 163:515, Feb. 9, e. Ohio M. J. 52:1306, Dec. 1956. 192. Hollister, L. E., Elkins, H., Hiler, E. C. ad. Sc. 67:789, May 9, 1957. 193. Hollister, L. [illegible] Stannard, A. N. and Drake, C. F.: 194. Holoubek, J. E., Thomas, O. C. and Sa[illegible]i, J.: Toxic reaction to 2-methyl- 95. [illegible] Karlish [illegible]). Canad. robamate (M[illegible]) in surgery of eye, ear, nose and throat. Eye, Ear, Nose & Throat. tranquilizer, meprobamate -- its use for pain associated with cancer, preliminary obamate (Correspondence). J.A.M.A. 161:644, June 16, 1956. 19[illegible] Kugelmass, I. N.: . P. Clinic 16:[illegible]; 1956. [illegible] Correspond- obamate in [illegible] surgical [illegible] 67:81[illegible] May 9, 1957. J. Chronic [illegible] 1956. [illegible] tranquilizing drugs. North-). J.A.M.A. 160:1431, April 21, 1956. 205. Lemere, F.: Habit-forming properties of 56. 206. Lemere, F.: Promazine (Sparine) in the treatment of psychiatric disorders. A. Arch. Dermat. & Syph. 74:437, March 1956. 208. [illegible] N. E. and [illegible], C. F.: Un- LoVan, P. [illegible] Wright, E. T.: Use of tranquilizers in diseases of the skin -- A Clinical evaluation of meprobamate in dermatoses and psychotic children. Ann. New .: Ineffectiveness of Dianox in the treatment of childhood epilepsy. Pediatrics 17: in treatment. Southwestern Med. 37:428, July 1956. 213. McG[illegible] R[illegible]: Drug reaction town in the treatment of chronically ill psychiatric [illegible]-patients. Pennsylvania M. J. ychiatric [illegible]se. W[illegible] 198[illegible] April 19[illegible] 21[illegible] Minos[illegible] M. G., Boyd, blication, 195[illegible] 217. Osinski, W. A.: Treatment [illegible] dietary states with meprobamate Osinski, W. A.: Treatment of anxiety states with [illegible]. New York Acad. spondence). J.A.M.A. 163:480, Feb. 9, 1957. 220. Pelner, L.: Meprobamate (Correspond- of six ataraxis in neuropsychiatric patients. Read at the American Psychosomatic robamate, [illegible] [illegible] cases, in 1957, pp. 25[illegible] [illegible] M. [illegible] ataraxics in on, V. M.: A two-year comparative study of ataraxics in neuropsychiatric patients. J. n (meprobamate) with psychotic patients. Am. J. Psychiat. 114:257, Sept. 1957. 226. 7. 227. Pennington, V. M.: Addiction to meprobamate questioned (Correspondence). North- in convulsive and related disorders. [illegible] 1012:1040, July 14, 1956. 229. Pevey, J. with phenobarbital, meprobamate and [illegible] agents. J. Pharmacol. & iltown) for the treatment of emotional disorders. Am. Pract. & Digest Treat. 7:1573, tion. North[illegible] [illegible] meprobamate in anx- 956. 233. Rushia, E. L.: Preliminary report on the use of meprobamate for pre-anes- S. B.: The effects of ward tension on the quality and quantity of tranquilizer uti- The value [illegible] muscle relaxants in disorders of muscle tone. Ann. New York Acad. Sc. Hypotensive [illegible] following treatment with meprobamate (Correspondence). Lancet 2: g drug: use [illegible] [illegible]). J.A.M.A. new tranquil[illegible] [illegible] J. Clin. & Exper. Psychopathol. 17:[illegible] March 19[illegible] 239. Sen- ode Island M. J. [illegible]:6[illegible] Dec. 19[illegible] [illegible] A. [illegible] Three cases O.: 'Mood [illegible] [illegible] [illegible]

Relieve moderate or severe pain

Reduce fever

Alleviate the general malaise of upper respiratory infections

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WITH

CODEINE PHOSPHATE*

maximum codeine analgesia/optimum antipyretic action

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...from pain of muscle and joint origin, simple headache, neuralgia, and the symptoms of the common cold.

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ACHROMYCIN® Tetracycline	125 mg.
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FORD, R. V., Rochelle, J.B.III, Handley, C. A., Moyer, J. H. and Spurr, C. L.: J.A.M.A. **166**:129, Jan. 11, 1958.

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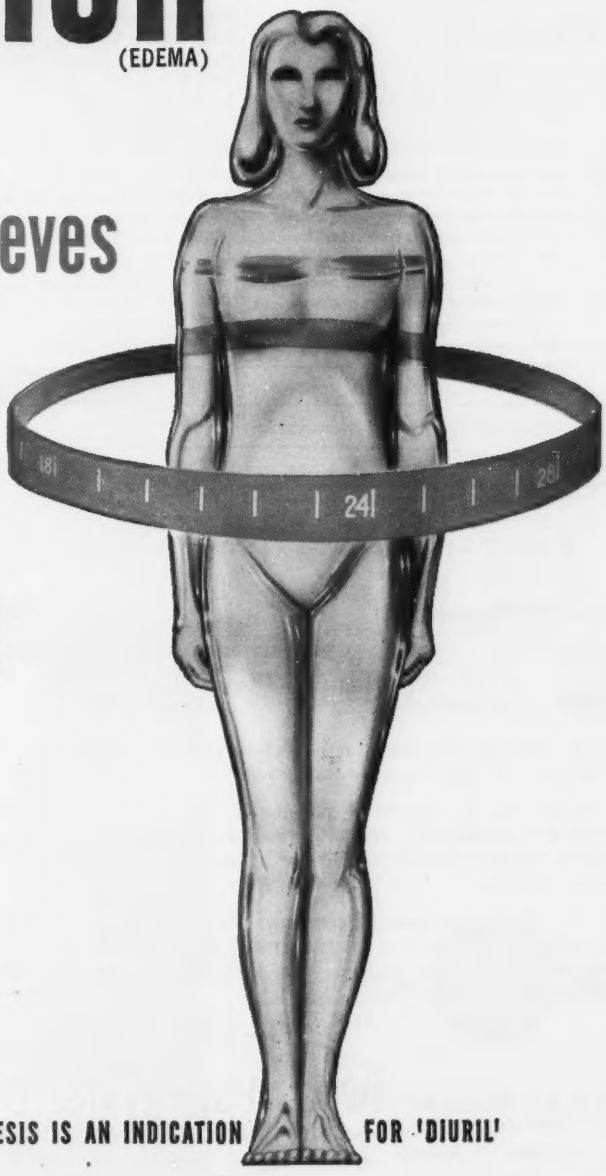

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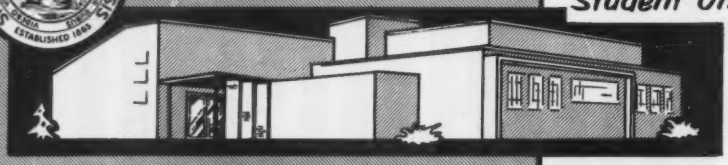

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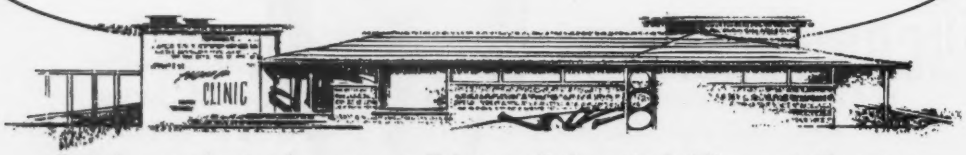

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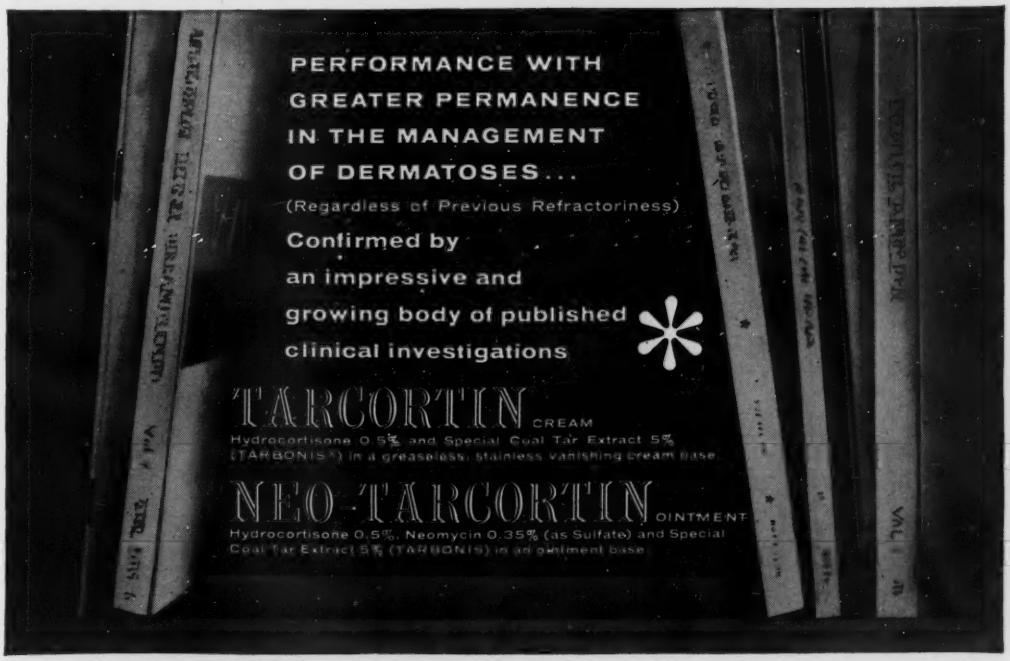
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1. Welsh, A. L., and Ede, M.: J.A.M.A. *166*:158, 1958.
2. Bleiberg, J.: J.M. Soc. New Jersey *53*:37, 1956.
3. Abrams, B. P., and Shaw, C.: Clin. Med. *3*:839, 1956.
4. Bleiberg, J.: Am. Practitioner *8*:1404, 1957.
5. Clyman, S. G.: Postgrad. Med. *21*:309, 1957.

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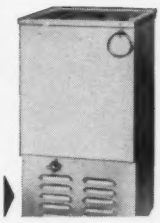

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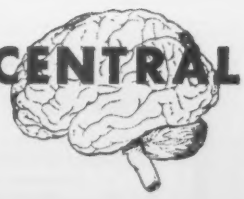

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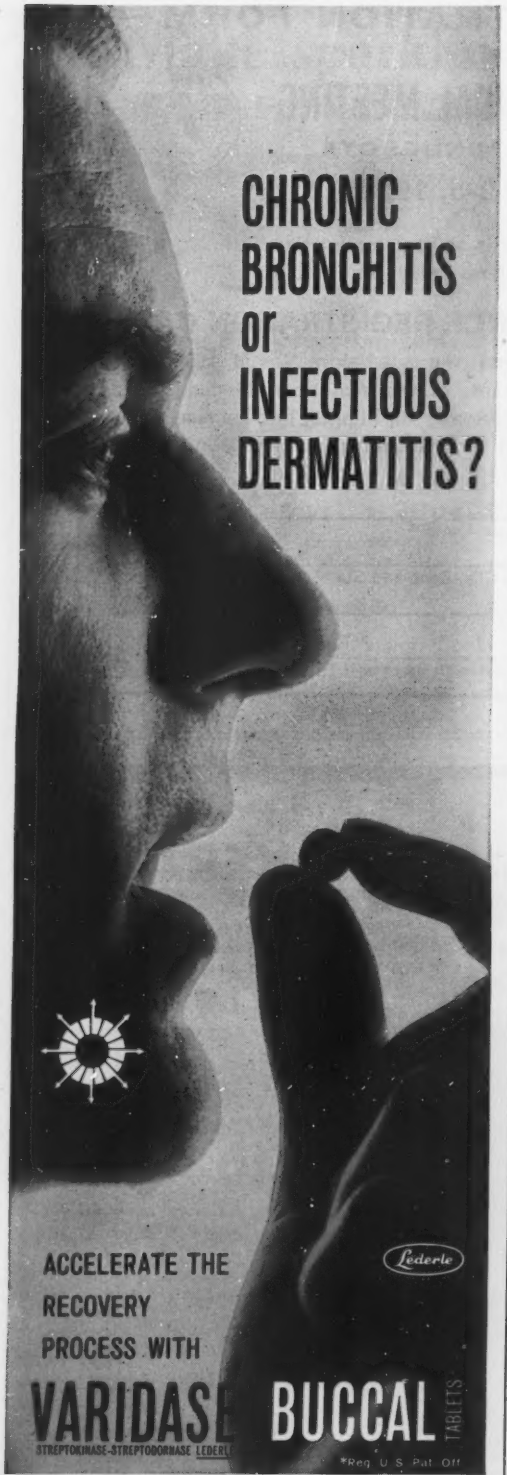

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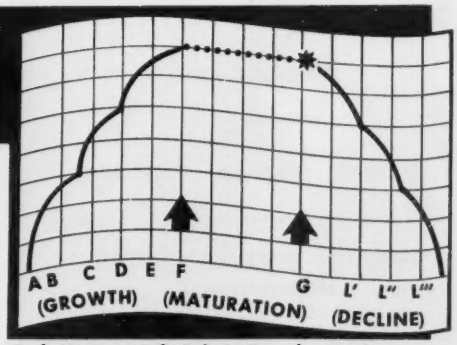

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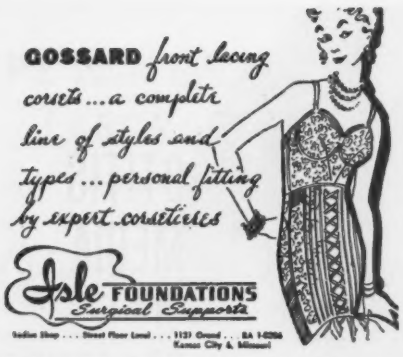
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Index to Advertisers

Abbott Laboratories ..... 17, 18, 19, 20, 21, and facing 16
American Medical Association 57
Ames Company, Inc. 30
Ayerst Laboratories 59

Burroughs Wellcome and Company, Inc. ..... facing 48

Chicago Medical Society 58
Ciba Pharmaceutical Products, Inc. ..... 9 and 26
Coca-Cola Company 54
Corn Products Sales Company 16

Farmers and Bankers Life Insurance Company 52

General Electric X-ray Department 56

Hanicke, P. W., Manufacturing Company 60

Isle, W. E., Company 62

Kansas Oxygen, Inc. 54
Kayser-Roth Company, Inc. 42

Lattimore-Fink Laboratories 60
Lakeside Laboratories 5
Lederle Laboratories 22, 25, 40, 49, 53, 58, 61, and facing 24
Lilly, Eli, and Company 28

Medical Protective Company 58
Menninger Foundation 53
Merck, Sharp and Dohme, Inc. ... 8, 14, 15, 33, 46, 50, 51
Munns Medical Supply Company, Inc. 60

Neurological Hospital 62

Parke, Davis and Company ..... Inside front cover and 3
Petro's Surgical Appliances 61
Pfizer Laboratories ..... 34 and 35
Physicians Casualty Association 53
Prairie View Hospital 61

Quincy X-ray and Radium Laboratories 61

Ralph Clinic 32
Reed and Carnrick 55
Riker Laboratories ..... Inside back cover
Robins, A. H., and Company, Inc. ..... 13, 41, and 47
Roerig, J. B., and Company, Inc. ..... 10, 11, and 38

Schering Corporation ..... 27, 44, and 45
Searle, G. D., and Company ..... 7, 29, and 55
Smith-Dorsey Company 39
Smith, Kline and French Laboratories ..... Back cover
Southwest Scientific Corporation 62
Squibb, E. R., and Sons 23
Sutliff and Case Company, Inc. 62

Tutag, S. J., and Company 59

University of Kansas Medical Center 52
Upjohn Company ..... 36 and 37

Wallace Laboratories ..... 6, 12, 31, and 48
White Laboratories 24
Winthrop Laboratories ..... 56 and 57
Wyeth, Inc. 43

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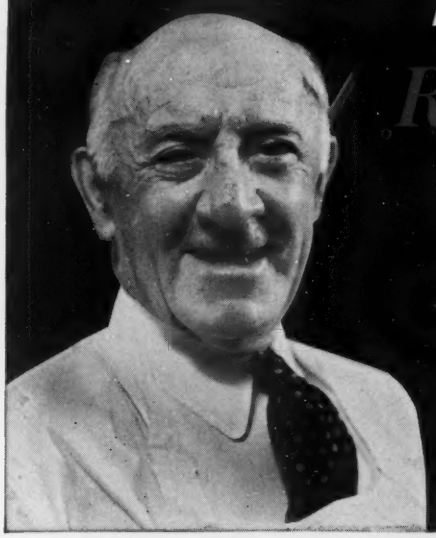

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Ford, R. V., and Moyer, J. H.: Rauwolfia Toxicity in the Treatment of Hypertension. Postgrad. Med. 23:41 (Jan.) 1958.

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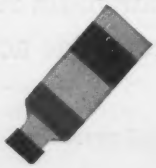

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